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Church Home and Hospital Issue*

THE CHURCH HOME AND HOSPITAL

THE CHURCH HOME AND HOSPITAL

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CHIEF OF STAFF

The original name of this institution was the Church Home and Infirmary. It is to be noted that the Church Home and Infirmary resulted from an amalgamation of the Saint Barnabas Church Home, on Biddle Street, and the Saint Andrew's Infirmary, on High Street; and on the

* The Church Home and Hospital Issue will be concluded in the April 1958 Maryland State Medical Journal.

day that the Church Home Infirmary was opened, in 1858, sixteen residents were brought to it from the Home and twenty patients from the Infirmary. The dual function of this institution has been continued, therefore, for one hundred years.

In its early days the Institution administered to the wants of the inmates and to out-patients who came in increasing numbers. Its beginning, like that of many successful institutions throughout our nation, was mediocre but it has grown and has served the community well during the first century of its existence.

In 1863 the report from the nurses mentioned the possibility of establishing a training school for nurses, "It is to establish there a Training School for Nurses where Christian women, who desire to devote themselves to such a calling, may find a home and gain the experience which is so necessary for its judicious and faithful exercise. This has been already done to a limited extent; but it is the wish of the Trustees to establish this as an integral part of their work. It is believed that such a central home for nurses would be a great blessing to the Home itself; to the women who might be instructed there, and to the community at large, who would thus be supplied with carefully trained and responsible nurses, whose fidelity can be vouched for by the physicians and other officers of the institution."

The year 1857 was a very important one in the history of the Church Home and Infirmary, for as it was in that year, as noted in the files of the "Sun" (week ending September 14, 1857) "For a Church Home—It is stated that several of the Episcopal churches of this city have purchased the Washington College building, on Broadway, with a view of converting it into a home for the destitute. It is well adapted for such an institution, having spacious grounds all around it."

The building mentioned above is the one in which the main entrance is located; the entrance leading into the familiar rotunda, above which the chapel is located—where church services are held at regular intervals. On the three floors above the chapel is the Home for Women, where many "dear old ladies" have spent their final years in comfort amid happy surroundings, and have been thoughtfully ministered to by the Clergy, the Woman's Guild, and have been carefully watched by nurses and physicians.

Then, in this year, Sister Adeline Blanchard Tyler was persuaded to come from Boston to take charge of the Church Home and Infirmary activities. She was indeed a remarkable woman in every respect. She was a conscientious worker, efficient, cooperative, and while young had mature judgment and had the ability to foresee into the future in a way experienced by very few people. Her interest in the sick and wounded parallels that of Florence Nightingale in the Crimea. Sister Tyler showed her ability in organizing, in the selection of a most satisfactory type of assistant that could be found, and of continuously improving the physical accommodations for her patients and domiciliary residents. Her ability to work with the representatives of her Church and with physicians, stamped her as a woman of remarkable but strong complacency. During the War between the States she showed her mettle when she went down to what is now Camden Station to visit some soldier boys from her home state of Massachusetts. It was against all rules and regulations for anyone to go in that area, especially a woman, but she accomplished her task and had some of the wounded transferred to her infirmary buildings, where she could give them personal attention, and where she could obtain the services of competent physicians.

It is interesting to note that the progress ever upward of the Church Home and Hospital parallels very closely the development of asepsis and antisepsis after Pasteur's great contribution. During the latter decades of the previous century, all hospitals had the same experience; in the summertime there were many cases of typhoid fever and malaria and in the wintertime

many respiratory diseases, although in those years more pneumonia patients were taken care of at home than in the hospitals. The amount of Surgery done gradually increased as Surgeons learned more and more about the technique of asepsis and antisepsis, but Surgery in this country did not take many forward steps until the last part of the last century and the first decade of this. With the discovery of x-ray by Roentgen, in 1896, the Church Home and Infirmary soon found it proper and convenient to establish an X-ray Department. Pathology was now being given greater consideration and the small department to house that facet of hospital practice was provided.

In 1907 a Committee, headed by Dr. Thomas S. Cullen, saw that the Church Home and Infirmary would provide a wonderful opportunity for the accommodation of more and more patients. His Committee interested a number of physicians both medical and surgical in the city, and they agreed to serve on the staff. From that time forward, which is one-half of the life of the Church Home and Hospital, progress has been made in all directions. The operating room facilities were totally inadequate, as were the laboratory facilities, both for the clinical laboratory and x-ray, and from time to time shifts were made until in 1922 the central building, one that was entirely modern for those times, was constructed. This made it possible to enlarge the suite of operating rooms and increase their number, to house the Department of Obstetrics, and to provide laundry and kitchen facilities for an expanded hospital. Less than thirty years later inadequacies of these expanded facilities were recognized and further improvements were made. An additional floor was built on the central building so that the X-ray and Cystoscopic Departments with expanded facilities could be located in this new area.

On October 1, 1912 Miss Jane E. Nash was installed as Superintendent of the Hospital and placed in charge of all administrative activities including those of the Nurse's Training School. A wiser choice could not possibly have been made. Miss Nash was an institution herself—she was well trained as a nurse and had spent several years in hospital administration in Salt Lake City, an experience she frequently recalled as being of great value to her in the ensuing years of service at Church Home and Hospital.

Her interest was total. The Nurse's Training School never could have been what it has been, had it not been for her vital concern in making it a training area, meeting the highest requirements of the nursing profession, and at the same time providing the highest degree of patient-care possible. Bedside nursing in this institution has been of such high order that it has been a cause for gratitude, as expressed by hundreds of patients and by many members of its professional Visiting Staff through the decades. Credit for this goes primarily to Miss Nash and the corps of assistants whom she chose.

Her interest in the Home was of the same high order. She knew every "member" of the Home. She visited them daily and was forever solicitous of their happiness.

As an administrator she enjoyed the cooperation of everyone with whom she came in contact. Her efficiency was outstanding, and she had the unqualified respect of The Board of Trustees, the Professional Staff, both nurses and physicians, and of the countless business associates which her position drew to her.

For more than fifty years the Church Home and Hospital has provided good training in both Medicine and Surgery. A large Visiting Staff, many of its members holding teaching positions of importance in either The Johns Hopkins or University of Maryland Schools of Medicine, has been in attendance, many of whom have been continuously available for, and interested in, instructing the Resident house staff. Many excellent Medical and Surgical Residents have gone out to all parts of the world to carry on as they were taught.

Church Home and Hospital marks its first century of usefulness to the community by entering its second with the full realization that progress must be continuous. There can be no let down. To meet the challenge of the second century The Board of Trustees has planned to build a much needed addition to the physical plant, and the Professional Staff has already extended the teaching program to meet modern demands.

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CHURCH HOME AND HOSPITAL has rich traditions which are unique to its history and past. One such tradition, the Morning Devotions, began almost at the original founding date of the Institution. At 7:00 each morning, the nursing staff gathers in the rotunda to hold a brief service which is piped by public address system throughout the hospital.

THE MEDICAL AND CHIRURGICAL FACULTY EXTENDS CONGRATULATIONS AND BEST WISHES TO THE CHURCH HOME AND HOSPITAL ON ITS CENTENNIAL CELEBRATION

This issue of the Journal helps to commemorate and direct attention to the Centennial Celebration of the Church Home and Hospital. Previously known as "Church Home and Infirmary," this institution has served an outstanding function in the medical annals of our community. It personifies and serves as a tribute to the courage, devotion and imagination of those who believe in the integrity of the individual patient in his relationship to his doctor.
—*The Editor*

IN MEMORIAM—GUY LE ROY HUNNER, M.D. 1869–1957

RHONA KUDER*

Dr. Guy Le Roy Hunner passed away on Sunday, July 14, 1957 at the age of 88. It is a sad thing to outlive one's nearest and dearest friends and colleagues for it leaves no one to write a tribute that can be worthy enough for so fine a man. He was indeed an eminent gynecologist and female urologist. In spite of opposition to some of the new ideas that he set before the medical profession, he labored on, stubbornly disregarding censure, and it was a great day for him when he received the coveted GOLD MEDAL for achievement in the field of urology from the Southern Medical Association.

He had nobility of character, geniality and a deep sympathy for all who needed his help. A quiet man, he would rather listen than talk, and few knew of his kindliness, of his love for art, music and the theatre, or of his charitable deeds. His goal in life was to one day retire to his farm on the Magothy River and read the books that he collected all through the years; but this was denied him for at about the age of 81 he developed cataracts. His hobby was his farm and he loved every blade of grass he coaxed out of the Anne Arundel sand. To see him working over week-ends, chopping wood, pushing the lawn mower, cutting down brush, one would not have thought that he would have energy left to come back to the city and

DR. GUY LE ROY HUNNER

* Miss Kuder was Dr. Hunner's secretary.

start a week filled with strenuous medical work,—but come Monday morning, he was as fresh and enthusiastic as ever for the week's tasks.

Patients, who by all the rules of the medical text-books should have long since gone to their Maker, are still living and well, due to his endless search for better and better ways of treatment. In this In Memorium encomiums are unnecessary as they are being expressed by these patients everywhere by telephone and letters. Such living tributes are more fitting than any that could be cast in bronze or hewn in stone.

There are many anecdotes that one remembers, which tell the character of our beloved Dr. Hunner, from the one when as a small Wisconsin boy delivering papers, he carefully placed each one out of the reach of snow or rain, to the adolescent, following the country doctor into the far countryside, bravely holding the ether cone and helping where he could. An unusual coincidence at the end of his college years helped to shape his outstanding career. On a camping trip in Yellowstone Park, he met some visiting German physicians, who, learning of his ambition to become a doctor, told him of the new Johns Hopkins Medical School about to be opened in Baltimore and about the rising young Dr. Howard A. Kelly, who was being brought down from Philadelphia to head the department of gynecology. From his camp site Dr. Hunner sent for application blanks and, having borrowed the money, he entered with the first class of the Medical School. That was money well invested.

Other stories come to mind,—he was the one-man audience of a Saturday night chamber music group in which Henry L. Mencken, Max and Elizabeth Brödel and four or five others were the active musicians. Although Dr. Hunner never played an instrument himself, he knew and loved music and this group loved having him present.

His kindness was a secret thing,—few, if any, knew of the hospital and nursing bills paid for from his own pocket, nor of the good food personally brought from his country place to coax back appetites of patients who had lost the will to live, nor of the patients invited to recuperate in his home before taking a train to a far-off home of their own. His family, too, shared in his kindliness; to him they brought their ill friends for "repairs" and recuperation. Even dogs found a way into their home to be nursed back to health.

Students and doctors on the brink of their careers came to him for advice. That he was a wise, and a patient counsellor can be attested by many now in the prime of their lives.

His was a simple life. He did not need monetary compensation for happiness; his greatest joy was a wife, children and grandchildren, whose lives filled him with love and pride. He was indeed a man who recognized his richness and thus acquired the greatest of all blessings, contentment. I knew him as few others did outside his own family and our association throughout the years has left memories surpassingly beautiful and given me abiding inspiration for the future.

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CHURCH HOME AND HOSPITAL

Rich in historical tradition, Church Home and Hospital still uses the original building purchased from the old Washington Medical College. Here, in 1849, Edgar Allan Poe died. Today there is a special commemorative window in the Institution to honor this Baltimore poet and writer.

Scientific Papers

PARASTERNAL THORACIC MASS

O. C. BRANTIGAN, M.D.*

The exact anatomic location of a thoracic lesion in the anterior pericardiophrenic angle, adjacent to the lower end of the sternum, with the mass extending posteriorly and laterally and blending with the heart shadow produces such a characteristic chest roentgenogram that the term, parasternal mass, is most appropriate (Fig. 1). The mass may be on either the right or the left side, but it is always more evident and more impressive when on the right. However, the possibility of the lesion being undetected on the left side must at all times be considered since it may be obscured by the heart shadow. The mass is always in intimate contact with the lower end of the sternum and anterior chest wall.

Though the position of the mass is characteristic and exact, the pathology that causes the mass is extremely variable[9] and is the result of (a) anatomic structural change, such as parasternal or Morgagni hernia, deformity of the diaphragm, congenital lobulation of the liver, and hypertrophy or other disease of the pericardial fat pad. (b) Neoplasm including a cyst. The neoplasm may be benign or malignant and may arise in the mediastinum, the lung or the diaphragm. The cyst may be inflammatory or congenital, originating in any of the locations mentioned. (c) An inflammatory lesion: the clear cyst, localized pleural pus pocket, localized pleural effusion, or a lesion of the lung parenchyma which would be located in the middle lobe of the right lung or in the lingular segment of the upper lobe of the left lung. (d) Cardiac abnormality: pericardial effusion, herniation of the pericardium, dilatation of the auricle, and ventricular aneurysm.

Since there is such a wide variety of pathologic causes for the parasternal thoracic mass, it becomes most important that an accurate diagnosis be made[9] because the proper handling of the patient will depend upon the diagnosis. In some patients with a parasternal mass no treatment will be necessary, for example, where there is a congenital abnormal lobulation of the liver (Fig. 2) or an enlarged pericardial fat pad. In other patients the pericardial cyst is adequately treated by simple aspiration (Fig. 3), whereas in the case of a neoplasm surgical excision is most important. The type of surgical approach may vary since the parasternal or Morgagni hernia containing bowel or without bowel can be repaired more easily through the abdominal approach and this operation will be better for the patient.

The diagnostic methods are merely the application of a group of standard procedures. By means of a complete history and physical examination signs and symptoms can be elicited that may suggest or eliminate a lesion in the lung parenchyma. If it appears that the lung parenchyma is involved, bronchoscopy, bronchography, and careful bacteriologic studies may be indicated. These measures are seldom required since the parasternal mass is rarely an involvement of the lung.

Roentgenographic examinations are important and, aside from the standard postero-anterior view, the lateral view is most essential since it will determine definitely whether or not the mass is in a parasternal location. Careful fluoroscopic examination of the parasternal mass is

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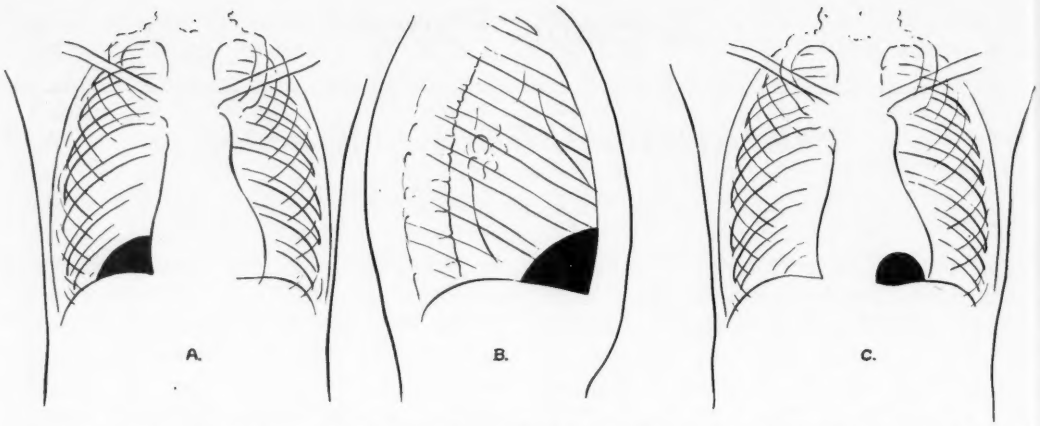

FIG. 1. A drawing showing the position of the parasternal thoracic mass. It is an easily defined and identified location in the pericardiophrenic angle adjacent to the sternum, extending laterally and posteriorly, and blending with the contour of the heart. To fit the criteria of parasternal mass it must be in intimate contact with the sternum, especially in the lateral view. This approximation to the anterior chest wall makes needle aspiration easy and safe. It may be the result of many different pathologic lesions.

always important in its identification. If air is seen in the mass it is certain to be a hernia. A barium enema will show whether or not colon is present in the mass or hernia (Fig. 4). A parasternal or Morgagni hernia may be on the right or left side, but rarely on both sides. If the parasternal mass is on the left side and does not contain colon, a study of the stomach may reveal it to be part of the hernial contents. When a diverticulum of the pericardium is suspected, roentgenographs taken in the Trendelenburg position may allow fluid to leave the cyst and enter the pericardium.[9] Other roentgenographic examinations, such as a planogram and angiocardiography may be helpful in rare instances.

If air from a pneumoperitoneum[2, 10] separates the parasternal mass from the diaphragm, it proves the mass to be either a hernia of Morgagni or a condition from below the diaphragm that is tenting the diaphragm around it. It could be a congenital lobulation of the liver (Fig. 2) or some other abnormality below the diaphragm. It must be pointed out that the failure of intraperitoneal air to enter into the mass does not completely rule out hernia or a lesion from the abdomen which is tenting the diaphragm upward, since there may be adhesions sealing off the upper abdomen.

The fourth important step in diagnosis is needle aspiration of the mass for fluid or tissue cells, that is, a needle biopsy.[4, 13] The intimate contact of the mass with the sternum and anterior chest wall makes needle puncture easy and reasonably safe. This procedure is most important if the history, physical findings, and roentgenogram have ruled out disease of the lung parenchyma and the presence of a hollow viscus in a hernia sac. A needle puncture of the colon or stomach, if these are contained in the mass, probably would not result in infection if covered by antibiotic protection. Needle puncture of a lung carcinoma may cause spread of the neoplasm in the needle tract but if given careful preliminary consideration the risk will be worth taking. If the mass is a pericardial cyst, the aspiration will be not only diagnostic but it may well be the only treatment that is needed[1] (Fig. 3). It will eliminate the necessity for a major thoracic operation. In the event of a neoplastic lesion the needle aspiration or biopsy often will prove diagnostic. Should the needle aspiration produce liver tissue and be com-

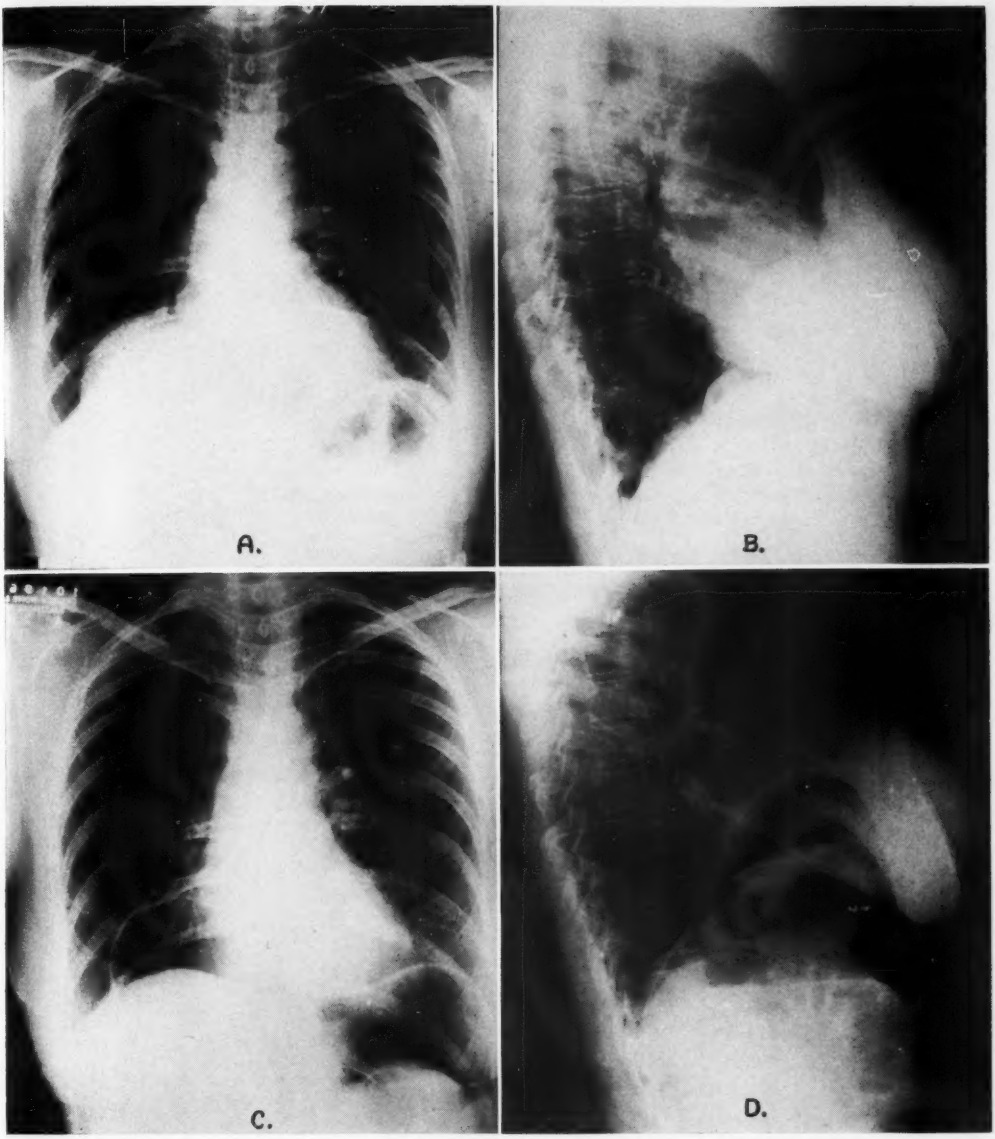

FIG. 2. These are roentgenograms of a white woman age fifty-eight years. She was free of symptoms. The abnormal roentgenographic findings were the result of a routine chest film. (A) and (B). The right parasternal thoracic mass is seen in typical postero-anterior and lateral roentgenographic views. The mass is located adjacent to the sternum. A needle puncture was done. Fluid was not found but the needle contents were saved for a biopsy specimen and revealed normal liver. (C) and (D). These roentgenograms were taken after 1000 cc. of air were injected into the peritoneal cavity. It will be noted that the air has replaced the liver that formerly occupied the area. The presence of normal liver was proved by needle biopsy. This is an instance of congenital abnormal lobulation of the liver. The patient required no treatment.

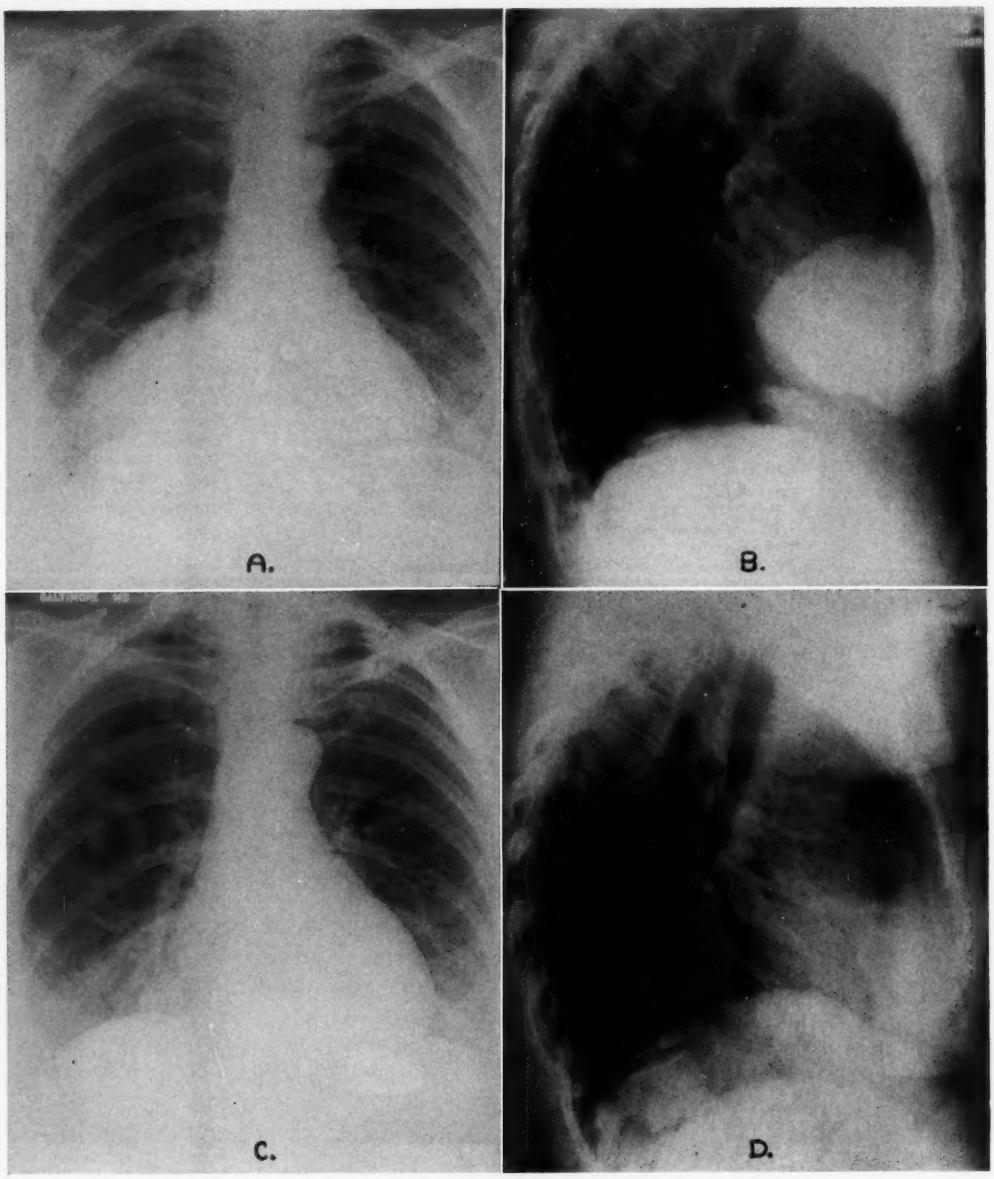

FIG. 3. Roentgenograms of a white woman, age forty-five years who had symptoms referable to chronic cholecystitis and cholelithiasis, which diagnosis was proved by cholecystogram and operation. The abnormal chest roentgenographic findings were the result of a routine chest film. There were no chest symptoms that could be elicited. (A) and (B). These postero-anterior and lateral chest roentgenogram views revealed a large parasternal thoracic mass which is directly adjacent to the sternum. (C) and (D). Roentgenograms of the same patient after the aspiration of clear, colorless fluid which revealed no abnormal cells and no bacteria after careful laboratory study. The diagnosis was pericardial cyst. No other treatment was necessary. The cyst has not refilled in twenty months.

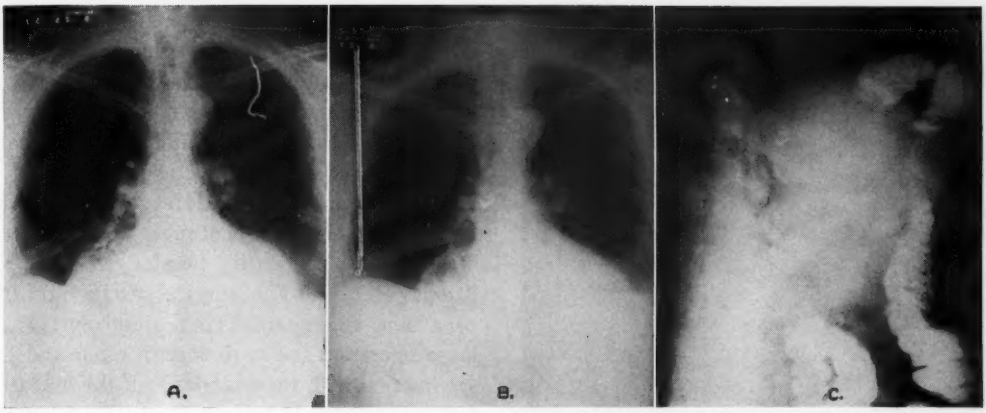

FIG. 4. Chest roentgenograms of a white male, age forty-three, who was free of all symptoms except indigestion, undoubtedly caused by cholelithiasis shown by cholecystogram. The chest roentgenograms were merely done on routine examination. (A) The illustration shows an air-containing parasternal mass. (B) In the mass the colon is identified by a barium enema. (C) The lateral view reveals a large amount of colon above the diaphragm in the parasternal thoracic mass, the parasternal or Morgagni hernia. The hernia was easily repaired by the abdominal surgical approach. The gallbladder filled with stones was removed at the same operation.

bined with pneumoperitoneum separating the mass from the diaphragm, it will prove conclusively to be an abnormal congenital lobulation of the liver (Fig. 2). It is conceivable that needle biopsy could reveal a liver tumor but the author has never encountered such a situation.

When the methods described are not adequate to make the diagnosis, pneumothorax[1, 2] can be employed. Its limitations, however, are noted in a fused pleura where air cannot be injected or in adherence of the lung about the mass so that a lesion of the lung parenchyma cannot be separated from any other type of mediastinal or diaphragmatic lesion. If pneumothorax separates the mass from the lung parenchyma it will not distinguish the type of the parasternal mass; however, such a mass can be identified by a closed thoracoscopic examination.[1, 10] Should there be a strong suspicion that the lesion seen on the chest roentgenogram is a hypertrophied pericardial fat pad (Fig. 5), pneumothorax and closed thoracoscopic examination would be the only method of diagnosis short of thoracic exploration.

When diagnostic methods fail to identify a localized chest lesion and also in some generalized thoracic or lung lesions, thoracic exploration is indicated as long as the physical condition of the patient will safely permit the operation. If the methods described herein do not identify the parasternal mass, thoracic exploration certainly is indicated. The discussion of diagnostic methods does not in any sense change the need for exploratory thoracotomy when the parasternal mass cannot be definitely identified.

It may be argued that prompt thoracic exploration with the application of corrective surgery will save time and in some situations even lessen the cost of patient care. Nevertheless, all surgeons abhor unnecessary operations. Logically the best surgical results follow the best preoperative diagnosis.

It is difficult to give a percentage figure, frequency or incidence of the various lesions comprising the parasternal thoracic masses. In a personal observation of more than 600 patients with carcinoma of the lung, the author has never seen one case that would fit the description of a parasternal mass. However, there is nothing to prevent its occurrence. Benign inflammatory lesions of the lung have been found

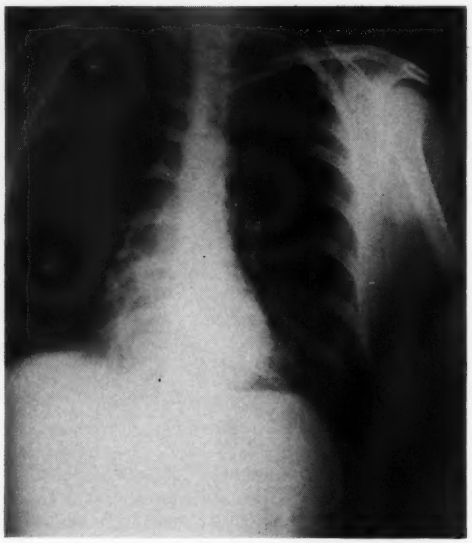

FIG. 5. Chest roentgenogram of a white woman, forty-one, who complained of chest pain and hemoptysis. Bronchoscopy and bronchography proved normal. A study of bronchial secretions and sputum revealed no evidence of malignant cells and no tubercle bacilli or other pathologic organisms. The chest film and fluoroscopic examination revealed the left-sided parasternal mass. Upon exploratory thoracotomy the mass was found to be a hypertrophy of the pericardial fat pad. After seven years the patient still has chest pain. For unexplained reasons hemoptysis has not been repeated. The lateral chest roentgenograms have been discarded and are therefore omitted.

in the parasternal area, particularly in tuberculosis and lung abscess. With a relatively large experience with mediastinal tumors, the author has yet to come across a single malignant mediastinal tumor in the parasternal area, although several patients with benign mediastinal tumors in the parasternal area have been encountered. In view of this experience there is little fear of spreading neoplastic cells in the needle tract by needle biopsy or aspiration. As one carefully considers the dangers of the spread of neoplasms, it is obvious that the risk is a good one if many patients can be spared a major operation.

Localized empyema and the localized collection of pleural effusion have occurred in the parasternal area. Needle aspiration will identify these conditions. Diaphragmatic tumors are relatively rare, only forty having been reported in the medical literature in all areas of the diaphragm.[3] In the experience of the author, the two lesions most commonly found to cause parasternal masses have been pericardial cysts[5, 13, 14] and the parasternal or Morgagni hernia. Yet, according to Hedblom[6] not more than 6 per cent of the diaphragmatic hernias are of the Morgagni type. The surgical excision of the usual and characteristic pericardial cyst was a large operation for an innocent lesion and dissatisfaction with the magnitude of the operation for pericardial cyst led to aspiration as a method of diagnosis and treatment. When a parasternal or Morgagni hernia is definitely diagnosed, the abdominal approach to its surgical correction is safest for the patient and technically easiest for the surgeon. The abdominal approach is almost imperative for the rare double or bilateral parasternal or Morgagni hernia. The author has seen only one of these cases.

Only one pericardial cyst and two parasternal or Morgagni hernias have been observed on the left side by the author. Ware[14] reported that in collected cases of pericardial cysts, forty-four were on the right side and seventeen on the left side. Harrington, according to Gudbjerg,[8] found only eight Morgagni hernias in 104 diaphragmatic hernias; of these, six were on the right side. Hypertrophy of the pericardial fat pad occurs most frequently on the left and is difficult to diagnose except by thoracotomy. One patient with hypertrophy of the pericardial fat pad on the left side has been operated upon (Fig. 5). However, it is obvious that pneumothorax with closed thoracoscopy will readily establish the diagnosis. This method will be used when such a condition is suspected again.

CONCLUSIONS

The parasternal thoracic mass is located in the pericardiophrenic angle adjacent to the lower end of the sternum. It extends laterally and posteriorly from the sternum, blends with

the heart shadow, and occurs more frequently on the right side of the chest. Pathologically it may be caused by a number of different lesions, of which pericardial cyst and parasternal or Morgagni hernia are the most frequent.

Methods of diagnosis have been described. If the parasternal mass is a pericardial cyst, diagnostic needle puncture will also serve as treatment. In the event it is a parasternal or Morgagni hernia, the abdominal approach is the best method of surgical correction.

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NITROGEN MUSTARDS IN THE TREATMENT OF CANCER

JOSEPH D'ANTONIO, M.D.*

In past years the treatment of cancers consisted of surgery, radium and x-ray singly or in combination, however, for these treatments to be successful it was necessary to stress the early detection of tumors in order to increase the survival of cancer patients. With the development of many of the carcinolytic drugs the inoperable cancer patient or the far advanced cases have been given some hope for relief from symptoms, pain, etc. and with some possibility of increased survival. A number of these chemical agents are quite familiar to all and are now available. This short discussion will attempt to bring together a few salient facts and features acquainting the practicing physician with their use.

The nitrogen mustard analogues are triethylene-melamine, mechlorethamine, nitrogen ethylene phosphormides and the triethylene phosphormides as well as triethylene thiophosphormides, chlorambucil and 14-dimethanesulfonoxybutane (myelran). Other agents that should be mentioned but not discussed are the Folic acid antagonists, aminopterin and amethopterin and also the purine antimetabolite 6-mercaptopurine. It is very interesting that the nitrogen mustards were investigated during World War II in secret under the control of the United States, British and Canadian Governments, by such men as Lindskog at Yale,

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J. Dougherty and Jacobson at the University of Chicago and Rhodes at Sloan-Kettering Institute. These studies were instituted because of previous knowledge gained during World War I when the Germans used the mustard gas successfully in combat. The highly irritating substance of the sulfur mustard gases showed not only local damage to the skin etc. but also serious systemic intoxication and dissolution of lymphoid tissues. In 1919 a pertinent observation was made that these gases produced a marked leukopenia and in some cases at autopsy an aplasia of the bone marrow with an effect on lymphoid tissue was found. Shortly after World War II the results of clinical studies with nitrogen preparations on many types of malignancies were published and soon afterwards the material in the form of mechlorethamine was made generally available. The nitrogen mustard or nitrogen analogues of sulfur mustard, retain their biological activities probably due to the beta chlorethyl grouping. From the reports of many investigators it is apparent that nitrogen mustard does not cure neoplastic diseases but as a palliative agent it is most useful in malignancies involving lymphoid and hematopoietic systems. There is no doubt that in the treatment of conditions known as lymphomas nitrogen mustard is most successful. It is also known that nitrogen mustard has a beneficial effect in the treatment of bronchogenic carcinoma.

Chemistry, Pharmacology and Cytotoxic Actions. It is important to remember that none of the compounds of the nitrogen mustard family in widespread use are specific cytotoxic agents only for malignant cells. The effects on normal tissues are often very severe and can be fatal. The nitrogen mustards are nitrogen analogues of the sulfur mustards used for gas warfare in World War I. The chemical activity of the compounds resides in the beta-chloroethylamine groupings, which are attached to nitrogen. In these compounds nitrogen has a valence of three and therefore a third substituant is also tacked onto the nitrogen atom. This substituant can be varied and accounts for the large number of substances which make up the homologous series. In the tertiary amine form the nitrogen mustards are not highly reactive, however, when placed in a neutral or alkaline aqueous solution, the structure undergoes transformation with a release of a chloride ion to form a cyclic ethylenimonium derivative, a quadrinary ammonium compound which is very highly reactive chemically. As soon as the ethylenimonium intermediate is formed, it can react with a large number of organic materials. After both chlorides are lost from the initial compound and the ethylenimonium derivative has reacted with the organic compounds in the body fluids, then it is no longer chemically reactive. Hence, the chemical activity in the body is extremely rapid making the reactions with biological compounds in the body very brief. However, the affects on the body and various portions of the body develop more slowly and are of longer duration. These few facts give some understanding of the actions and the toxicity of the drug. This is also the reason why, clinically, before administering nitrogen mustard, it is necessary to rapidly place the material in solution and inject it into the subject immediately.

It is generally believed that the mechanism of the cytotoxic action of alkylating agents, such as the nitrogen mustards, is related to its ability to combine with many organic radicals of biological interest and importance. It is a known fact that the toxicity and inhibition caused by the mustards on cells occurs in a premitotic phase of cell division. In experimental material such as amphibian embryos, and in experimental animals, lethal doses of mustards seem to affect all germinal tissue such as bone marrow, lymphoid tissue, as well as the epithelium of the cornea and intestinal mucosa. Embryonic cells which normally differentiate into non-proliferating adult tissue undergo fragmentation and disintegrate. However, cells which differentiate into permanently germinal tissues such as lymphoid tissue, undergo cyto-

plasmic hypertrophy and differentiate into more adult types without further mitosis. This is also seen in higher forms of life. In humans, the more differentiated elements of bone marrow and lymphoid structures will disintegrate very rapidly after administration of nitrogen mustard. Many of the cytological actions may well be explained upon the high re-activity of the mustards with nucleic acids. Apparently, the nucleic acids of the desoxyribose type seem to be more readily inactivated by these agents. Many of the actions of the nitrogen mustards are very similar to those of ionizing radiation from x-ray. Phillips in his reports published in 1950 demonstrated many similarities between the biological actions of these two agents. Because of this similarity, many of the actions of nitrogen mustard are known as radiomimetic.

Absorption, Fate and Excretion of the Mustards. Nitrogen mustards can be absorbed from the gastrointestinal tract, however, this often causes many severe local reactions in the lining of the intestine. For this reason, an intravenous route is by far more practical. A few minutes after injection into the body, the mustard undergoes a chemical transformation to an inactive form, as stated above.

Although germinal tissue is highly susceptible to the actions of nitrogen mustard, when using the compound clinically, the only normal tissues which are significantly affected are the bone marrow and lymphoid organs. The effects on lymphoid tissue have been observed as early as 10 hours after injection of nitrogen mustard fragmentation of the tissue and atrophic changes being shown. With the usual therapeutic doses, the bone marrow has a definite but rather protracted course, changes are usually manifested by the 4th day becoming maximal by the 10th day. This is temporary and regeneration occurs usually within 2–3 weeks. About 24 hours after therapy the depression of the lymphocytes is evident and this progresses for 6–8 days. After a few days, granulocytopenia occurs and this lasts from 10–21 days. Thrombocytopenia may be seen and may be severe. A drop in the red blood count may be seen for the first few weeks, however, this drop is not as dramatic as the effect on the white blood count. There is no doubt that the severity of changes produced by mustard is directly related to the amounts given. Fortunately, the depression of hematopoiesis that is seen with the use of therapeutic doses is a reversible one. Shortly after administration of the agent and its analogues both orally and intravenously, varying degrees of nausea and vomiting follow within 1–3 hours. This is probably related to the fact that the germinal epithelium of the intestinal mucosa is highly susceptible to the actions of the agent. The gonads of humans are also susceptible and in females amenorrhea of several months duration may follow a course of therapy. Autopsy studies by Spitz have shown impairment of spermatogenesis in males after treatment with mustard. The action of nitrogen mustards on proliferating cells is of a nonspecific nature and this probably is the reason for the dramatic affects sometimes seen on normal tissues as well as in malignant lymphomas and in undifferentiated bronchogenic carcinomas, the latter being the only epithelial cell cancer in which the mustards seem to be effective to any degree.

Therapeutic Uses. Some 40 or more compounds with nitrogen mustard activity have been studied. Of these the bis,beta-chloroethylamine hydrochloride or the HN2 with the trade name of mustargen has received widest use. This compound is given intravenously. The substance when dissolved in aqueous solution or saline must be administered very promptly because the activity is lost very quickly when in solution. With an intravenous saline solution running, it is best to inject the material into the lumen of the tubing to decrease the possibility of thrombophlebitis of the vein and prevent extravasation into subcutaneous tissues. The usual course consists of a total dose of 0.4 mg. per kilogram of body weight divided into 4

equal doses and given on consecutive days. Many variations have been adopted depending on the inclination and experience of the therapist. In the treatment of bronchogenic carcinoma the total dosage has been between 0.6–0.8 mg. per kilogram of body weight. This is a highly dangerous dose and frequently it is associated with marked reaction and death. With a large dosage the patient's course sometimes resembles the delayed death syndrome seen when experimental animals are given minimal lethal doses of nitrogen mustard. There is a definite sequence of events with anorexia, vomiting, hemorrhagic enteritis, diarrhea, polyuria, loss of electrolytes and intracellular potassium, shock, coma and respiratory failure. Animals and also patients with symptoms resembling this syndrome do not respond to vigorous supportive therapy comprising replacement fluids, blood transfusions, etc. Nitrogen mustard may be repeated and responses may or may not be successful, but repeated courses should not be given until the bone marrow and peripheral blood have returned to normal levels.

Treatment Using Mustargen (Nitrogen Mustard)[1]. There is no doubt that the nitrogen mustards have a specific field of usefulness in the treatment of Hodgkin's disease. Other diseases such as lymphosarcoma, chronic leukemia and bronchogenic carcinoma also show response. However, they have not shown the dramatic response of Hodgkin's disease. When Hodgkin's disease is generalized with diffuse lymphadenopathy, hepatomegaly, splenomegaly and intrathoracic extension, nitrogen mustard therapy is more efficacious than radiation and often life-saving since radiation would cause edema, swelling and perhaps encroach on the respiratory tract. With mustard therapy, the action is immediate, quicker and more generalized causing reduction in the size of the involved lymphoid tissues and without dangerous edema. There is no doubt that at times the response of the disseminated Hodgkin's disease to mustard therapy is very gratifying with reduction in fever within a few days, return of the appetite and strength and when anemia is present, often improvement of the anemia. The duration of the induced remission is variable and may range from a few weeks to many months. Often, these remissions become progressively shorter after repeated doses of nitrogen. The stage in which the disease is treated also affects the remissions and the results obtained by therapy. However, when disseminated Hodgkin's disease has become resistant to roentgen therapy the effects achieved with nitrogen mustard are often very gratifying and at times appear complete. There is no doubt that nitrogen mustard has contributed much to the management of this disease even though the life span of the patient has not been lengthened considerably.

In the treatment of the lymphosarcomas, there is no doubt that the giant follicular sarcoma gives the most gratifying response while small cell or lymphocytic lymphosarcoma and reticulum cell lymphosarcoma has a rather poor response. The life saving possibilities of nitrogen mustard in the treatment of these diseases when they have involved vital areas, is worth-while mentioning. Although there are other alkylating agents available for the treatment of leukemias, the nitrogen mustards may also be used. There is much greater success with chronic myelocytic leukemia than with other types of leukemia. As stated previously, bronchogenic carcinoma is the only epithelial cell tumor that responds to this form of therapy. The rapidly progressive bronchogenic carcinoma such as the "oat cell type" seems to be the most responsive. In actual use, however, it has been rather disappointing and difficult to evaluate.

Treatment Using TEM (Triethylene-melamine)[2]. There are many compounds with nitrogen mustard activity being used by investigators. Among the more useful is triethylene-melamine which is converted from a

[1] Made by Sharp and Dohme (Merck).

[2] Made by Lederle Laboratories.

less to a more active state in an acid medium (quadrinary ethylenimonium compound) and thereby exhibiting chemical properties similar to other nitrogen mustards. Although the actions of TEM are delayed, it has the definite advantage of causing less anorexia, nausea and vomiting and of allowing oral administration. In the acid media of the stomach, the change to the active form is rapid and absorption is unpredictable. The administration of sodium bicarbonate at the same time allows for the more predictable transformation and absorption of this drug. It is usually given on awakening in a dose of 5 mg. in two successive days along with 2 gm. of sodium bicardonate. This is the usual treatment for most cases of Hodgkin's disease. Often, when the desired effect has not been obtained, an additional 5 mg. may be given two weeks later. The lymphosarcomas and chronic lymphocytic leukemias seem to be particularly sensitive to this drug. Remissions can be obtained with approximately half the dose used in Hodgkin's disease. As in the case of many forms of treatment, many investigators have introduced various regimes that differ quite widely in dose and time. Maximal depression of the bone marrow occurs in approximately ten days with TEM. Once a remission has been induced, most therapists wait for a relapse in the disease before resuming therapy. Along with the fact that TEM can be given orally there is less nausea and vomiting associated with treatment. The main disadvantage is the delayed onset of action, which is probably related to its poor absorption and the need for transformation from the less active form to the more active form of ethylenimonium compound.

Treatment Using Myleran (14 dimethanesulfonoxybutane)[3]. In attempting to evaluate biological activity of simple sulfonic acid esters, the British in 1953 synthetized a nitrogen mustard-like substance known as myleran; a long chain chemical known as 14-dimethane-sulfonoxybutane. Chemically, it bears very little resemblance to nitrogen mustard; however, its activity is similar due to the fact that it is also an alkylating agent. Although the cytotoxic action of myleran is not entirely understood its activity is unique in that it selectively depresses the normal and abnormal myelocytic tissues. The fact that its toxic action does not extend to germinal cells and lymphoid tissue in gastrointestinal epithelium as in nitrogen mustard makes it a much more desirable chemotherapeutic agent in the treatment of myeloid leukemia. The drug is well absorbed from the gastrointestinal tract and it is very effective after oral administration. The drug has been successfully used by many investigators in the treatment of chronic myelocytic leukemia. Many therapists feel that this is the chemotherapeutic agent of choice and probably on a par with X-ray therapy and radioactive phosphorus. Wintrobe reports favorable results in the treatment of 16 patients. Spurr et al. reported highly favorable results in the treatment of 11 cases of chronic granulocytic leukemia. He felt that within the controlled therapeutic range it was of considerable value and certainly on a par with effects of X-ray radiation and radioactive phosphorus. In this series of cases, the total dosage of myleran varied from 244 mg. to over 3,000 mg. The average daily dose was approximately 3.2 mg. with a range from 2–8 mg. per day. The single dose was given before breakfast by mouth with very little gastric discomfort. The drug should be continued under very close supervision with frequent hematological studies until the white blood count is between 10,000 and 20,000 per cubic mm. Successful treatment is associated with improvement of appetite, gain in weight, a sense of well being and a decrease in leukocyte count. The white count may begin to decrease between ten days to two weeks, although at times it may not be apparent until the third or fourth week of therapy. If anemia is present it is often relieved. Quite often in chronic lymphatic leukemia,

[3] Made by Burroughs Wellcome & Co.

there is a very high platelet count and this is often reduced with therapy. The only important side effect of the drug when used in therapeutic dosage, is thrombocytopenia; however, in the cases reported in the literature this is easily controlled and does not appear to progress to thrombocytopenic purpura. The platelet reduction is usually slower than the granulocytic decrease. The development of uricemia from the rapid destruction of the granulocytes with deposition of urates in the urinary tract has been mentioned but this has been a very uncommon occurrence. It has been reported that resistance to myleran does develop, but the drug has been used with success following resistance to X-ray therapy. Investigators seem to differ as to whether patients should be carried on maintenance doses or be maintained on intermittent therapy with the onset of relapses.

Treatment Using Lukeran (Chlorambucil)[4]. Recently a newer nitrogen mustard derivative having a formula very similar to the parent substance known as chlorambucil, formerly known as CB 1348, has been made available. It was developed in attempting to find a mustard-like material as effective as mustard but without the need for intravenous administration and the troublesome side effects. In a course of study, by Bouroncle, et al. of 42 patients of which 24 had Hodgkin's disease treated with chlorambucil, the therapeutic effects were considered good on Hodgkin's disease but not with acute monocytic leukemias. Far advanced chronic lymphatic leukemias previously treated with radioactive phosphorus and X-ray therapy showed no clinical or hematological improvement. One case of reticulum cell sarcoma showed a very favorable response; and one case, lymphosarcoma, showed no improvement. Acute lymphatic leukemia occurring in a four year old white boy was treated unsuccessfully. There was also very little success with mycosis fungoides and multiple myeloma. Ulpmann, et al. felt that the greatest value of the material was in the treatment of chronic lymphocytic leukemia, especially when the patients with this disease had a moderate thrombocytopenia. They did not feel that patients with lymphocytic lymphosarcoma or Hodgkin's disease showed as good a response.

They further felt that response to nitrogen mustard or triethylene-melamine in these two diseases were superior to chlorambucil. Patients in this series received oral doses ranging from 5–30 mg. daily for 5–7 weeks so that the average course amounted to approximately 350 mg. As in the case with myleran, the cytotoxic effect of chlorambucil appeared to be selective mainly for lymphocytes. This substance has shown to be only partially radiomimitic producing chiefly the lymphoid effects of X-ray radiation. In human subjects, absorption through the intestinal tract is good; however, in rather large doses nausea and vomiting can be produced. With regular therapeutic doses, the effects on the bone marrow are only moderate and seem to be reversible. Patients who have lymphomas appear to be more sensitive to the drug and smaller doses are needed. It is quite possible to produce a severe neutropenia and thrombocytopenia with overdosage. The usual oral dosage is from 0.1 to 0.2 mg. per kilogram of body weight ranging in duration of 3–6 weeks. It is not necessary to divide the daily dose. The maintenance dose must be carefully graded according to the response of the patient and the hematological picture. It has been reported that patients with Hodgkin's disease seem to need a slightly larger daily dose than those with lymphoma or chronic lymphocytic leukemia. It is important that the patient be followed as in the use of all these substances, and that weekly hemoglobins, hematocrits, leukocyte counts and differentials be done. The patient should be seen at least once a week. Lymphopenia may be slowly progressive and often a neutropenia is seen by the third week of treatment, sometimes continuing for ten days to two weeks following the last dose. The response and effect on the blood seems to be related to the size of the dose and the length of treatment. In treating these patients over a long period, chemotherapy

[4] Made by Burroughs Wellcome & Co.

should not be carried out at the same time as radiation therapy. A study of the bone marrow should be performed on all patients and the bone marrow should be without infiltration of lymphomatous tissue and appear within normal range before beginning treatment.

Treatment Using Tepa and Thio-Tepa (thiethylene thiophosphoramide).[5] The N-Ethylene phosphoramides (N - N′ - N″ - Triethylene - Thiophosphoramide) are nitrogen mustard-like substances which for investigational use only and are only available through special permission from the Lederle Laboratory Division of the American Cyanamid Company. A number of workers have reported the use of these substances in practically every known malignancy. It has been reported to be of some value in cases of malignant melanoma, neuroblastoma, Hodgkin's disease, carcinoma of the breast and some of the undifferentiated carcinomas. It is of particular interest in that it can be given safely by the intravenous, subcutaneous, or intramuscular route, or directly into the tumor mass. A few investigators have reported that this material can also be given into the spinal canal and into brain tissues, without irritation or necrosis. The compounds are usually employed as a one per cent aqueous solution; however, the material must be sterilized by Seitz filtration without the use of any heat and must be used within a period of approximately ten days and kept refrigerated and away from light. This solution has been used after a much longer storage period on a patient with Paget's disease of the breast, with good results. Many investigators advise that the dosage not be greater than 50 mg. for any single course and that the amount administered be in the proportions of 5–10 mg. per day until the tolerance of the patient is ascertained. There is considerable variation in the total amount used and also the duration of therapy. It is unusual for any one patient to tolerate more than 75–100 mg. of this material in a short period of time. Occasionally, there is evidence of bonc marrow depression but not usually with therapeutic doses. Peripheral blood counts and platelet counts should be done at least once a week. With tepa, the platelets may appear more sensitive and drop slightly; however, with the use of thio-tepa this is not often seen. When the white count or platelets begin to drop, the medication should be stopped and the patient watched for delayed depressive effect.

[5] Made by Lederle Laboratories.

Conclusion

The practicing physician should be familiar with the various nitrogen mustards that are being used in the treatment of carcinoma patient.

The physician by the use of these agents can add great comfort to the patient and perhaps lengthen the survival period.

A few salient facts and features concerning the nitrogen mustard-like substances are presented in the hope that practicing physicians might make this type of therapy available to the patient.

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HYPERPLASIA AND TUMORS OF BRUNNER'S GLANDS OF THE DUODENUM

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Hyperplasia and tumors of Brunner's glands are comparatively rare. The impression is also gained from a perusal of the literature that the incidence of Brunner's glands tumors among benign tumors of the duodenum is relatively low. Jacobius[13] in 1940 reviewed the literature, and found 17 cases of Brunner's glands adenoma of the duodenum, adding a case of his own. Wilensky[20] in 1948 found 46 recorded cases, to which he added another. Hudson and Ingram[12] in 1952 collected 65 cases. Deren and Henry in 1956 found 70 cases and added a case of their own. Since Hudson and Ingram's report in 1952, only 8 additional cases are recorded.[1, 3, 5, 6, 7, 16, 17] Up to 1957 there were 73 recorded cases of Brunner's glands tumors of the duodenum. Most of these were single case reports.

Brunner's glands are acino-tubular glands found in the deep layers of the mucosa and in the submucosa of the duodenum. Embryologically, they appear about the sixth month, as massive epithelial ingrowths in the depths of the duodenal crypts.[15] These glands are located largely in the submucosa, being most abundant in the proximal portion of the duodenum and diminish in number distalward toward the duodenojejunal junction. According to Landboe-Christensen, Brunner's glands do not extend beyond the ligament of Treitz. However, in rare instances these glands may extend into the proximal portion of the jejunum, and are not infrequently observed in the pylorus. Robertson had directed attention to the marked similarity of the cells of Brunner's glands to those of the pyloric glands in both appearances and staining reaction, which led to the assumption that the former are a direct continuation of the latter. The chief difference is that Brunner's glands break through the muscularis mucosae, whereas the pyloric glands are wholly confined to the deep layers of the mucosa. They are also distinguished from pyloric glands by their large size.

Feyrter[9] studied 2,800 duodeni, classified hyperplasia of Brunner's glands into three types, namely, (1) diffuse hyperplasia, (2) circumscribed nodular hyperplasia, and (3) adenoma, an isolated growth composed of localized hyperplasia. Robertson grossly examined 15,000 duodeni, and microscopically examined 1,000 cases and described the pathology of Brunner's glands under five headings, namely, (1) congenital malformation, (2) atrophy and hypertrophy, (3) degeneration and infiltration, (4) inflammation, and (5) hyperplasia and neoplasm. He pointed out that complete absence of the glands and other anomalous irregularities has not been observed, and that the actual quantity of Brunner's glands vary within wide limits. According to Wilensky, the neoplastic changes, because of its essentially benign nature and the fact that the structure reproduces the glandular architecture of the normal gland, must undoubtedly have dominating relationships to the functional activity of the gland rather than a true neoplastic mechanism. This seems to be borne out by the frequency with which cystic disease takes place. In 2 of our collected cases the adenoma was associated with cystic changes, apparently indicating a similarity of the pathologic process. It would seem that the various pathologic changes involving Brunner's glands, i.e. hyperplasia, hypertrophy, adenoma, and cystic formation are varying degrees of a similar pathologic process. Other investigators have expressed similar views.[1, 5, 14]

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The majority of reported cases of Brunner's glands tumors were adenomas. The tumors may be single or multiple, sessile or pedunculated. Adenomatous tumors are more apt to be single. The hyperplasia type occurs more or less diffusely, involving either a localized segment, or the entire duodenum. The duodenal bulb is the most common site of Brunner's glands tumors. The size of the growth varies from 0.2 cm. to 6 cm., and in rare instances it may be much larger. In most cases there are no other associated pathologic conditions. In a few instances it had produced an intussusception. When the pylorus is involved, it may cause obstructive signs.

There are many gaps in our knowledge of the physiology of Brunner's glands. It has been definitely established that these glands excrete a continuous alkaline mucoid secretion. Florey and Harding[10] believe that the alkaline mucus secretion tends to protect the duodenal mucosa from damage by acid gastric juice. The possibility that malfunction of the secretory action of these glands might lead to the development of an ulceration has been considered by some authors.

The etiology of the various pathologic changes occurring in Brunner's glands has not been fully established. In spite of the marked activity of these glands, the vast majority of them show little evidence of being pathologic. There is a general agreement that the incidence of pathologic changes occurring in Brunner's glands are minimal, and this is attested by the fact that comparatively few cases of Brunner's glands pathology are recorded. Its occurrence in a few cases in children,[4, 19] and in 1 case in a stillbirth[8] lends support to the possibility that there may be a congenital etiology in some cases. Hartz and van der Sar[11] believe that in the presence of acute inflammatory conditions, the epithelium of Brunner's glands react in the same way as other glandular epithelium. They examined 24 duodenums, 8 of which revealed a proliferative activity. According to Hudson and Ingram, accumulated evidence has shown that chronic inflammation may predispose the glands of Brunner to hyperplasia with resultant adenoma formation. On the other hand, Robertson[18] examined 1,000 duodeni microscopically, found that the outstanding characteristic of the glands is their comparative freedom from influences which ordinarily affect secretory glands, and that in the presence of acute inflammatory conditions of the duodenal mucosa, even with erosions or ulceration, the Brunner's glands are not especially involved.

Brunner's glands tumors may occur at any age, from infancy to old age, most often between the 4th and 6th decades. They occur predominantly in the male sex.

Brunner's glands hyperplasia and tumors may be asymptomatic. The symptomatology, if it occurs, is vague and not pathognomonic. In some instances the condition may produce symptoms requiring medical treatment and often necessitating surgery. When the condition involves the pylorus, it is apt to cause symptoms. The following symptoms have been encountered in the recorded cases, namely, pain, unrelated to food or soda, though in a few instances there was food or soda relief. An excessive amount of gas, identified by bloating, belching, and flatulence was a prominent symptom. Nausea and vomiting occurred frequently. Bleeding was present in a few instances when it was complicated by ulceration. Gastric analysis was mentioned in 9 of the 35 cases. Of these, 6 revealed a hyperacidity, 1 a hypoacidity, and in 2 there was no free acid. Cases with hyperplasia of Brunner's glands are apt to be associated with hyperacidity. In 5 cases of hyperplasia, 3 presented a hyperacidity, and in the other 2 cases no gastric analysis was made. A preoperative diagnosis of Brunner's glands tumefaction has been made in some instances. In most recorded cases the diagnosis was made post-operatively or at autopsy. The roentgen examination offers the only means of demonstrating a pathologic condition, and a correct diagnosis can often be made, especially if the

condition is sought after. When the tumefactions are small, multiple and diffuse, the diagnosis of Brunner's glands hyperplasia may be suspected, while the single polypoid type simulate other polypoid growths.

Summary

Brunner's glands tumors of the duodenum are comparatively rare. The etiology, physiology, and pathology of Brunner's glands tumefactions are discussed. There are no pathognomonic symptoms characteristic of this condition. The diagnosis of Brunner's glands pathology should be suspected in all cases of tumefactions involving the duodenum. Although the roentgen diagnosis of Brunner's glands tumors has been made in many instances, the criteria are not characteristically pathognomonic. However, the occurrence of multiple and diffuse tumefactions produced by hyperplasia should lead one to suspect the involvement of Brunner's glands.

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PHYSICAL FACTORS RELATING TO RESPIRATORY COMPLAINTS

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Any patient's complaint must first be approached from the standpoint of what has occurred to alter the normal activity of the area involved. This, of course, implies that one must first understand the normal physiology of the area in question and the factors affecting it. In this instance, I shall limit the discussion to certain histologic and physiologic factors concerning respiratory mucosa and the physical factors of temperature and humidity.

It is well known that the normal respiratory mucosa is a pseudostratified ciliated columnar epithelium which in the nose runs from the sinuses and preturbinal areas backward to the choana where it fades into the transitional epithelium of the nasopharynx; and from the subglottic area into the tracheobronchial tree. Covering the respiratory membrane is a mucus coat, which under optimum conditions, is propelled from the sinuses and nose posteriorly by the intrinsic motion of the cilia. Under these same conditions the ciliary beat is from 8–12 times per second and the speed of the mucus stream 0.25–0.75 centimeters per minute. This varies in different areas. The significant conclusion to be drawn from these facts is that the entire mucus blanket of the nose is discarded in the nasopharynx at least once every half hour.

The same general conditions hold true for the tracheobronchial tree, where the direction of flow is from below upward toward the pharynx.

In addition to the above factors, it is also known that the nose acts as a filter, warms the inspired air and humidifies it. Regarding the nose in its role as a filtering mechanism, we can immediately correlate the mucus coat as a cleansing mechanism. This would mean that, under optimum conditions, matter removed from the inspired air would rapidly be discarded on a continuous conveyor belt into the nasopharynx, from whence it would be conducted downward in the alimentary tract by the swallowing act or expectorated. Ciliary activity, however, is impeded or lost by drying, heavy metal salts, cocaine, epinephrine, water, other agents and disease.

The nose also has a marvelous ability to warm the inspired air, for, practically speaking, regardless of the temperature at the nostril, the temperature at the choana is 97–98 degrees Farenheit. This fact is of particular significance and will be referred to later.

As regards humidification, the degree of saturation of inspired air by the nasal mucosa has been variously estimated from 75–95 per cent relative humidity. For the preparation of this air, about 680 cc. of water must be supplied by the respiratory tract for pulmonary consumption during a 24 hour period. The amount of water supplied by the respiratory tract, however, varies considerably with the temperature and humidity of the atmosphere.

The majority of the above information may be found in, what I consider to be a classic in the field of Otolaryngology, Applied Physiology of the Nose, by Arthur W. Proetz.

Correlation with Complaints

Although all of the aforegoing information is fairly common knowledge among our profession, little attempt has been made by most to apply it. It must be noted here that the conditions referred to above relate to optimum physiological conditions as regard the individual, the absence of local disease and an environment

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in which the temperature is at 72°F. and at least 45 per cent relative humidity.

A moment's observation will recall to mind the fact that most respiratory complaints and infections occur from fall through spring, while, on the other hand, the warm humid weather of summer is relatively free of such complaints. This would seem to correlate with our understanding of normal nasal physiology since cold air is dry air, especially in so far as the human respiratory tract is concerned. Drying, as has already been pointed out, impedes or stops ciliary activity. The obvious result is a slowing or stoppage of the cleansing action of the respiratory membrane. Carrying these thoughts a little further, we also know that the body is constantly in combat with the bacteria in its environment. Certainly great assistance is lent to the bacterial flora of the nose when there is slowing or stoppage of ciliary action, since the bacteria are then given adequate time over a cell membrane to secrete their enzymes, break down the membrane and make their entrance. In response to the bacterial accumulation caused by the filtering action of the nose and impedance of the cleansing mechanism, the body responds in the next best manner in which it can; it calls up more blood to bring white cells and antibodies as protective measures. This is congestion!

Let us now consider a nasal passage which is 4 millimeters wide at its widest point. Suppose on each side we now add 1 millimeter of congestion. It is plain to see that we have cut the airway at least in half. If we now add 2 millimeters of inspissated secretion, we have nasal congestion or obstruction. Consider now the effect on the much smaller airways of an infant or child!

Certainly with impediment of the mucus flow, there is increased absorption of the bacterial flora which must be neutralized in the lymphoid accumulations of the head and neck, such as the adenoids, tonsils and cervical nodes. It follows, that with increased absorption of the bacterial flora over a period of time, there will be lymphoid hypertrophy. It would seem reasonable to assume, therefore, that the most common focus of infection in the nose and throat that results in lymphoid hypertrophy is the inspissated, secretion coated mucus membrane.

It is not uncommon, however, when explaining to a patient that low humidity is their problem to have them respond, "But, doctor, I have my worst difficulty when it is damp and foggy or raining!" This is undoubtedly true, for as indicated earlier the nose is an exceptionally fine apparatus for warming the inspired air. We must now examine the term "relative humidity" and realize that it is simply the amount of moisture that air contains at a given temperature compared to what it could hold when completely saturated at that temperature. The temperature is, of course, the key to the matter. Damp air is cool air and although saturation may be 100% at the nostril, the warming incurred as air passes to the choana causes it to expand and the relative humidity to drop.

As an example, let us say that we have atmospheric air at 50 degrees Farenheit and 100 per cent humidity and let us heat this air to 98 degrees Farenheit. In order to maintain the same 100 per cent humidity, and using the figure of 500 cubic feet of air exchanged every 24 hours by an average individual, 488 cc. of additional water would have to be supplied by the mucous membranes every 24 hours.

However, 100 per cent atmospheric humidity occurs infrequently during fall, winter and early spring. The more frequent occurrence of 40 degrees Farenheit and 20 per cent humidity would require an expenditure of 602 cc. of water every 24 hours to maintain a level of 98 degrees Farenheit and 100 per cent humidity.

In view of the above information it is evident that the warming of inspired air is a drying process as regards mucous membranes.

In addition it must be noted that the above examples refer to an atmospheric level continuous for 24 hours. Under average daily conditions, however, night air is usually cooler and drier than day air. Furthermore, there is a fluid balance deficit during the sleeping hours which

occurs due to the lack of fluid intake during this period; thereby resulting in increased inspissation of secretion.

Another common complaint which may be correlated with drying and inspissated secretion is that of post nasal "drip." This complaint is most common in the morning and at night, although, it may be present throughout the day. The majority of these patients complain of, "a lot of post nasal drip." Actually, examination will usually reveal no increase in volume of post nasal drainage but rather a reduction in volume with a change in character of the secretion to a more viscid and tenacious quality. In fact, it is this quality which makes the patient conscious of his post nasal discharge.

Finally, it is understandable that with drying, accumulation of bacteria and the chronic infection thereby set up, there will be a degree of omnipresent congestion which will result in making worse or initiating other conditions such as eustachian obstruction, laryngitis, pharyngitis and sinusitis.

It is not intended here to convey the impression that all cases of nasal congestion, post nasal discharge, sinusitis, pharyngitis, eustachian obstruction, and tracheobronchitis are due to drying. Some of these, of course, will be caused by allergy, others by acute infections, vasomotor rhinitis and other specific conditions. Nevertheless it is important that the physical factor of drying be not overlooked in the treatment of respiratory conditions.

Treatment

Obviously it is impractical to treat nasal congestion by advising the patient to stay in a humidity of 75 per cent at 72°F. Therefore, the approach has been to advise patients suffering from the minor complaints of drying to use an 8 hour vaporizer in the bedroom at night since this is the driest period of the day for the human body as well as the atmosphere. (During the sleeping period water is lost with each expiration but is not replaced until the patient awakens. In regard to the atmosphere, because most of us sleep at night, there is no sun to warm the air and increase moisture content. This is especially noticeable during the change of seasons when the days are warm and the night cool; the moisture at that time of the year may be seen to condense on cars and other objects during the night and disappear as the sun reappears on the following day.)

It is not enough, however, to simply advise a patient to use a vaporizer. He must also be instructed in its use. In this respect, the object is to trap the warm humidity by closing the doors and windows to the room; then, if the room is later found to be too stuffy for sleep a window or door may be opened just sufficiently to make sleep possible.

During the day, if nasal drying is a problem, a plastic "spraypak" filled with normal saline may be used as a nasal wash, by spraying the nose periodically, drawing the solution through and expectorating it, several times until the nose feels clean.

Of course, if the condition is an acute one and precludes normal daily routines, it is reasonable to invoke 24 hour treatment with a vaporizer.

Although the information contained herein has been aimed primarily at the nose and nasopharynx it must be understood that the same principles hold true as pertains to lower respiratory conditions such as chronic laryngitis, chronic bronchitis and some chronic coughs and atelectasis.

It is the simple fact that this fairly common condition frequently goes undiagnosed and is treated with repeated courses of antibiotics, antihistamines and what have you before being recognized that has caused me to write this article. The following are a few examples of such cases.

Case # 827

This 5 year old boy was first seen on January 11, 1957 complaining of chronic cough with expectoration of phlegm for a period of 7 months. There was a history of nocturnal cough and chronic nocturnal fever averaging 1–1½

degrees. Four months prior to his first office visit he had developed pneumonia and was treated with penicillin and tetracycline. He also complained of occasional sneezing and had been treated with an antihistamine.

Examination revealed fluid behind both tympanic membranes, hypertrophic tonsils and adenoids and moderate cervical adenopathy. A smear of the nasal secretion was negative for eosinophiles.

Treatment consisted of the nocturnal use of a vaporizer.

Within one week the patient was asymptomatic as regards his cough and fever. There has been no recurrence. His aural condition was later relieved by adenoidectomy.

Case # 170

This 52 year old, white male was first seen on October 11, 1955 complaining of post nasal discharge for 3 years or more and gradually becoming more noticeable. He stated that his nasal congestion was worse in the morning and evening and that his nose was frequently obstructed on the right.

Examination revealed the nasal membranes to be congested and swollen, there was moderate congestion at the sphenoid rostrum and over the soft palate, oropharynx and hypopharynx. A tenacious mucoid secretion was present in the nasopharynx.

A diagnosis of nasopharyngeal drying was made and an eight hour vaporizer advised at night. He was also advised to use normal saline as a nasal spray, as necessary, during the day.

The patient was not seen again until March 1956 when he returned complaining of a head cold and nasopharyngeal soreness. He stated that he had obtained good relief with his vaporizer during the preceding months until just the past few days. He was asked to continue the use of his vaporizer and was placed on warm saline gargles in addition. The latter to be used only until the soreness had ceased.

He was seen once again in April 1956 complaining of sore throat. This was during a period in which he had been doing considerable smoking. He was asked to cease.

It is to be noted that the patient never complained of post nasal drainage or nasal obstruction following the first visit. He has remained free of complaint until the present time.

Case # 18

This 6½ year old boy was seen in March 1955 with the complaint of recurrent earaches for the past 5 years. These attacks had been associated with fever and antibiotics had been required for treatment. Sore throats had occurred infrequently.

Examination revealed the right tympanic membrane to be injected and there was no light reflex. Neither tympanic membrane moved on Toynbee test. A large amount of dried secretion had to be removed from the nose before examination could be made. A moderate size adenoid and chronically inflamed tonsils were present.

A diagnosis of eustachian obstruction due to chronic infection was made. The nocturnal use of an eight hour vaporizer was advised.

The patient's father was last seen in June 1957. He stated that the vaporizer had been used with good results and that the child had had no further difficulty.

Summary

The essential facts concerning respiratory mucus membranes, their physiologic requirements in regard to ciliary activity, moisture requirement and the effects of temperature and humidity upon them have been discussed. In addition, these facts have been related to some common respiratory complaints and examples have been given.

It is the purpose of this essay to emphasize that consideration of physiological requirements

be given respiratory problems before proceeding to other forms of therapy.

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THE PRIMARY MALABSORPTION SYNDROME

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INTRODUCTION

A primary Malabsorption Syndrome may be defined as a state in which there exists impairment of absorption of numerous food elements, accompanied by striking metabolic disturbances, in the absence of demonstrable organic disease of the gastrointestinal tract, liver, or pancreas. This classification covers those entities previously termed as Tropical or Non-Tropical Sprue, Childhood and Adult Celiac Disease, and Idiopathic Steatorrhea, which are believed to be clinical varieties of the same genetically transmitted metabolic derangement.

PHYSIOLOGY

Impairment of absorption from the small intestine remains the major obvious abnormality. Manifestations of an active malabsorption state may begin in early childhood or remain in a latent status until advanced adulthood. The terms latent and active describe this abnormal state very well, as it is often characterized by remissions and exacerbations. Numerous environmental factors, such as: emotional upsets, pregnancy, fatigue, improper diet, tropical and non-tropical infections, etc., have been regarded as capable of converting a patient with latent malabsorption into an active or manifest state.

The impairment of absorption affects all organic food principles including carbohydrates, proteins, fats, calcium, phosphorus, sodium, potassium, chloride, iron, Vitamin C, Vitamin B complex, Vitamin B12, Vitamins A, D, E, K, carotene, Folic Acid, and even water. Prolonged loss of these necessary increments cause multiple signs and symptoms. Selective malabsorption may occur in some instances without clinical recognition of other underlying abnormalities. Incomplete clinical pictures present difficult diagnostic problems. Occasionally an isolated symptom (hypoalbuminemia and intractable edema) may dominate the clinical scene.

Decreased absorption of fat leads to diarrhea, steatorrhea, and weight loss; the excessive stool fat carries with it calcium combined as soaps and fatty-acid complexes. Failure to reabsorb calcium from the pancreatic and intestinal juices also appears to be an important factor in the calcium depletion. Fat-soluble Vitamin D is also lost contributing further to the calcium

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deficit. Eventually, hypocalcemia and latent or active tetany will result. Poor absorption of phosphorus plus the lowered calcium will cause osteomalacia, with generalized demineralization, bone pain, real and pseudofractures. Decreased absorption of carbohydrates leads to abdominal distension, flatulence, and a flat glucose tolerance curve. Loss of other fat-soluble vitamins as Vitamin A and carotene will cause low plasma levels and a flat Vitamin A tolerance curve. A lack of Vitamin K will produce a hypoprothrombinemia and hemorrhagic complications. Lack of normal iron absorption may lead to an iron deficiency anemia. Poor absorption of Vitamin B12 and Folic Acid may produce a macrocytic anemia with a megaloblastic marrow. Vitamin B complex deficiency may cause glossitis, stomatitis, cheilosis, and peripheral neuritis. Hypoproteinemia, hypoalbuminemia, edema, and ascites may also occur. Increased protein may be lost in the stool, if steatorrhea is very heavy. However, the profound protein deficiencies probably depend more upon inadequate intake and impaired synthesis. Hyponatremia and hypocalcemia with dehydration, hypotension, and acidosis are common. Even water metabolism is disturbed in this syndrome. There has been shown to be a delay in the excretion of water with abnormally long retention within the small bowel.

All these aberrations demonstrate that the small intestine of these patients acts like a flabby, distended tube with resulting inefficient transport and absorption of fluids and foods.

Clinical Manifestations

Females seem to be affected slightly more often than men. As stated, the disease can become active in early childhood or in any later decade. Patients with latent malabsorption states have no appearance of illness. However, patients with activity present a completely different picture. They will complain of diarrhea, foul-smelling, watery or mushy yellow-gray stools, often bulky, occurring 5–10 times or more per 24 hours. Quite common complaints are nocturnal diarrhea and diuresis. Gradually, they develop abdominal distension, gaseousness, abdominal discomfort, flatulence, and loss of weight, sometimes of remarkable amounts. Glossitis and stomatitis will usually develop. Eventually there will be some loss of appetite although good appetite may remain despite the weight loss. Hemorrhagic complications may occur varying in severity from skin and mucous membrane petechiae, to generalized ecchymosis, and gross bleeding from the gastrointestinal and genito-urinary tracts. All stages of muscular irritability may be found, and occasionally tetany will be the main presenting symptom. Bone pain or a pathologic fracture may be quite prominent features.

Physical examination during this stage will show a chronically ill patient with obvious weight loss, general muscular wasting, dehydration; areas of dark brown pigmentation are occasionally found over the forehead, cheeks, dorsum of the hands, and extensor surfaces of the forearms. Examination of the mouth will show a red, smooth tongue. Hypotension and a sinus tachycardia are present. Pitting edema of the legs will be observed. The most striking physical finding is generally the abdomen. Despite the frailness of the remainder of the body, the abdomen is full, distended, and protuberant. To palpation it is soft, doughy, and non-tender. Loops of intestine can be felt. Prominent tympany can be elicited. Evidences of bone pain can be discovered. Clubbing of the fingers may be present. Decreased vibration sense may be noted.

Laboratory Features

Hematology

A macrocytic anemia is the most frequent finding, often it will be associated with a megaloblastic bone marrow. A hypochromic, microcytic anemia is found most commonly in those patients who have a history of childhood mal-

absorption. Leukopenia and thrombocytopenia are not uncommon.

Blood Chemistry

Hypoproteinemia, hypoalbuminemia, hypocalcemia, hyponatremia, hypokaliemia, hypophosphatemia, hypoglycemia, hypocholesterolemia, hypopholipidemia, hypolipemia, hypoprothrombinemia, and elevated alkaline phosphatase may be discovered in the full-blown state.

Gastric Analysis

Most often a hypochlorhydria is found, but achlorhydria can occur as part of the Malabsorption State.

Absorption Tests

A flat glucose tolerance curve is a prominent feature. Serum carotene and vitamin A show reduced to low serum levels. The vitamin A tolerance curve is likewise flat.

Pancreatic Studies

In nearly every instance the Secretin Test will show a normal volume and bicarbonate response.

Fecal Studies

Stool collections will usually reveal a steatorrhea of over 10 per cent of the ingested dietary fat. Values as high as 50–60 per cent may occur in very active malabsorption states. No excess of protein is generally found, unless a marked steatorrhea is present.

Vitamin B12 Absorption Test

Vitamin B12 is believed to be mainly absorbed in the distal small intestine. Decreased absorption of radioactive Vitamin B12 is usually determined by finding a reduced excretion into the urine after a loading dose. Also serum Vitamin B12 levels are reduced.

Roentgen Findings

Studies of the gastrointestinal tract have shown the most characteristic changes in the appearance of the small intestine; that is, dilatation, segmentation, fragmentation, hypersecretion, moulaging, mucosal modifications, and motility differences.

Dilatation is the most frequent and impressive alteration, particularly in the mid- and distal jejunum. Usually the most pronounced dilatation is found in the more advanced cases. The mucosal folds can be coarsened, thickened, and at times, even obliterated.

Segmentation refers to the clumping of barium in large, widely-separated, elongated masses, usually in the mid- or distal ileum. Two forms of segmentation have been described by Marshak et al., immediate and delayed. Most commonly delayed segmentation occurs in the ileum several hours after ingestion of the barium meal. In severe cases, segmentation is often immediate, occurring as soon as barium enters the small intestine. This phenomenon seems to bear a relationship to increased intestinal fluid content.

Fragmentation refers to thick, coarse, mottled remnants of residual barium which coat the mucosa after the main barium column has passed to other loops. Normally, only a fine, faint, stippling of barium remains.

Hypersecretion is demonstrated in several ways in addition to its role in segmentation. In a normal small intestine, barium has a clean, homogenous character, which in malabsorption states is replaced by a coarse, mottled appearance. Air-fluid levels will occasionally be noted because of the excessive secretion present throughout the entire small intestine.

The "moulage sign" refers to a smooth tube-like appearance in which all mucosal markings are obliterated.

There is considerable variation in the transit time. Most often travel through the small intestine is slightly prolonged, ranging from three to seven hours. Rarely a marked hypermotility will occur.

Roentgen studies of bones may show evi-

dences of generalized demineralization, deformities, pseudo-fractures, and true fractures.

Special Diagnostic Study

A method of accomplishing duodenal and jejunal biopsies by means of blind suction technique through a special plastic, flexible tube has recently come into prominence. Biopsies from the duodenum and upper jejunum have shown distinct mucosal changes. The main abnormalities have ranged from blunting and reduction of the villi to their almost complete atrophy. The surface epithelium and the glands of Leiberkuhn have shown atrophic changes. This method of study should help to bring about better understanding of this unusual syndrome.

Therapy

General Measures

Proper physical and emotional rest are extremely important in the management of the Malabsorption patient. During "inactive" periods, sufficient sleep should be obtained. An "active" state can be caused by overwork and fatigue. Sensible emotional attitudes should be encouraged. Prolonged anxiety and tension can result in the activation of symptoms. With any overt sign of activity, bed rest is indicated.

Infections are also frequent offenders in the renewal of active malabsorption. Any acute infection should be treated early and vigorously. During any infectious state, dietary control should be watched more closely.

Diet

Although the fundamental defects responsible for this syndrome are obscure, satisfactory regulation has been possible by dietary management. Classically these patients have been maintained upon a high-protein, high carbohydrate, and low fat diet (30–70 grams). Fruit juices, bananas, and strawberries have been advised in larger quantities because fructose absorption is normal. If dietary fat is properly restricted, gradual relief of symptoms will generally result.

In patients who are not well-controlled by dietary measures alone, steroid therapy may be instituted with remarkable effectiveness. The usual initial dose of Cortisone is 100–150 mgs., or of Meticorten 50–60 mgs. Gradually, this dose is tapered by weekly reductions until it is no longer necessary, or until the smallest maintenance dose compatible with relief of symptoms is found. Uninterrupted use of the steroid may be needed for prolonged periods or indefinitely. Even patients who have successful results with steroid therapy must continue some limitation of fat in the diet.

In recent years excellent claims have been reported for the gluten-free diet, particularly in many childhood Malabsorption patients. The gluten-free diet eliminates all foods containing wheat and rye flour. Frazer has demonstrated by fractionation of gluten, a glutamine-containing peptide which may be responsible for the postulated harmful effects of the gluten diet. The proper role of the gluten-free diet awaits further long-term clinical experience.

Drugs, Vitamins, and Minerals

For the prevention and/or treatment of the macrocytic anemias, periodic intramuscular injections of crude liver extract, Vit B12, and the oral or parenteral use of Folic Acid are necessary. In iron-deficiency anemias, the new intramuscular iron-dextran complex (Imferon) is very effective if oral iron therapy is inadequate and whole blood not essential.

Deficiencies of Vitamin B complex and Vitamin C can be prevented by taking 1–2 of these capsules daily. Potassium and sodium supplements can be given to prevent dehydration and lassitude. Vitamin K should be taken in order to keep the prothrombin time at a safe point. Hypocalcemia requires large doses of calcium salts orally; during latent or active tetany, additional calcium must be given intravenously, as well as increased amounts of Vitamin D. Osteomalacia may also be helped by increased parenteral dosage of Vitamin D and testosterone. There are oral water-soluble prep-

arations of the fat-soluble vitamins (other than K) available for maintenance use.

Differential Diagnosis

A number of disease entities can occasionally simulate the Primary Malabsorption State. These have been called Secondary Malabsorption States and include: Lymphomata of the small intestine and mesentery, Amyloidosis, Intestinal Lipodystrophy, non-specific jejunoileitis, chronic pancreatitis, mucoviscidosis, carcinoma of the pancreas; after surgery, as sub-total gastrectomy, inadvertent gastro-ileostomy, and extensive small intestinal resection. In most instances the resemblance to the Primary State ends with the finding of steatorrhea. Except for the extensive small intestinal resections or gastro-ileostomies, tetany, osteomalacia, macrocytic anemia, as well as most of the other laboratory features (flat glucose tolerance and Vitamin A tolerance curves) occur only in the Primary Syndrome. An occasional case of lymphosarcoma has defied early diagnosis. Generally, however, differential diagnosis between the two states should not be difficult.

SUMMARY

The Primary Malabsorption State is believed to be a genetically transmitted metabolic derangement. Impairment of absorption of all food principles, vitamins, electrolytes, water, etc., from the small intestine remains the major obvious aberration, and leads to multiple signs and symptoms.

Laboratory examinations will disclose widespread abnormalities. The most characteristic roentgen changes will occur in the appearance of the small intestine. A method of obtaining duodenal and jejunal biopsies through a special tube by a blind suction technique has become available as a clinical tool.

The classical diet consisting of high-protein, high carbohydrate, low-fat diet is still in use. Often it is supplemented by steroid therapy. In addition the gluten-free diet is getting careful clinical surveillance.

The differential diagnosis between the Primary and Secondary Malabsorption States should generally offer little difficulty.

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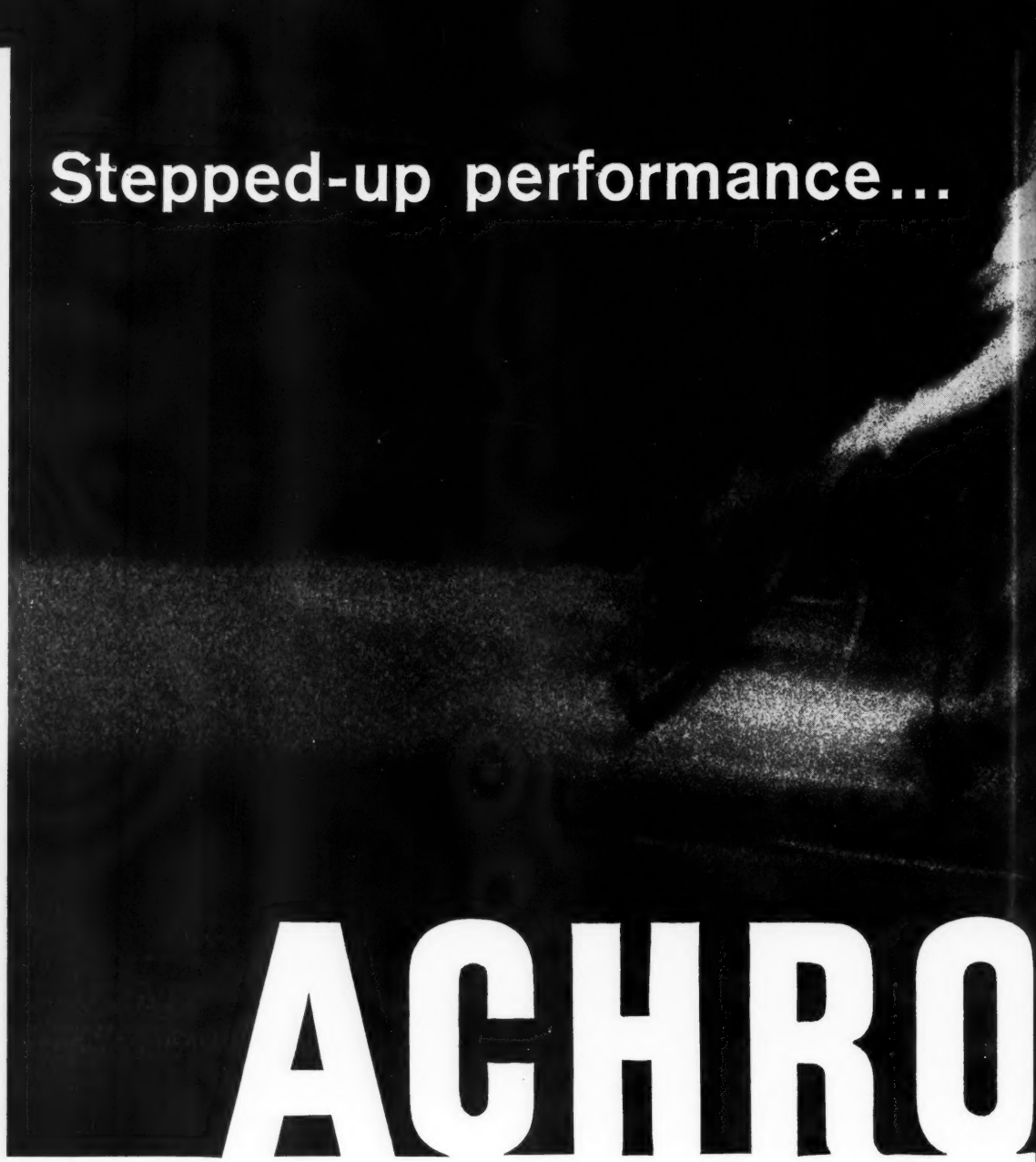

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AMENORRHEA AND INTERSEXUALITY

HOWARD W. JONES, JR., M.D.*

General Considerations

The realization that the fundamental genetic sex of an individual may be readily and simply determined by cytologic examination of suitable material such as buccal (figs. 1 and 2) or vaginal smears has demonstrated that a small but significant group of women who find their way to the practitioner or gynecologist and who complain of amenorrhea, are in fact genetic males. A few of these women—and the word women is used advisedly to indicate their social sex—may be immediately suspected of their hermaphroditic state by anomalies of their external genitalia and their masculine habitus. Others, however, are entirely feminine in every way including their secondary sexual characteristics, so that the discovery of their intersexuality often comes as a surprise. It is the purpose of this paper to call attention to male intersexuality as a cause of amenorrhea by

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cataloging those forms of the disorder which have amenorrhea as a prominent or presenting complaint. It is obvious that only patients who are regarded as girls and women and who are above the age of puberty will be considered. Furthermore, female hermaphroditism due to adrenal hyperplasia will, likewise, not be considered although it is important to recognize

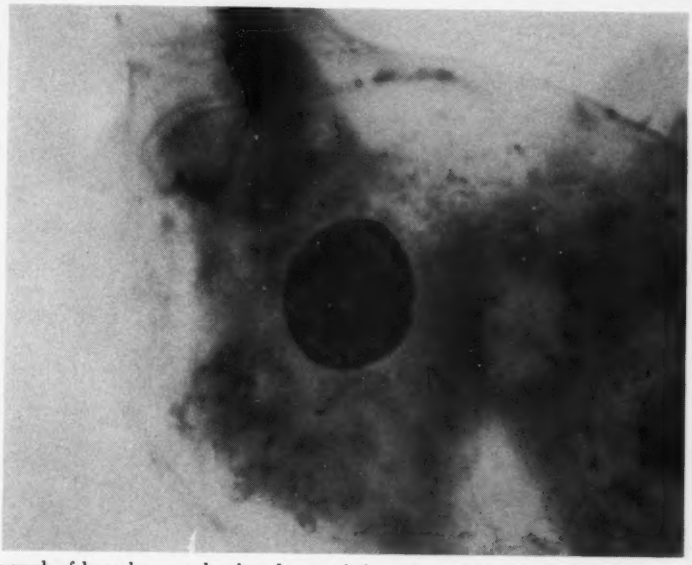

FIG. 1. Photomicrograph of buccal smear showing characteristic nuclear body subjacent to the nuclear membrane (Papanicolaou stain, × 2000).

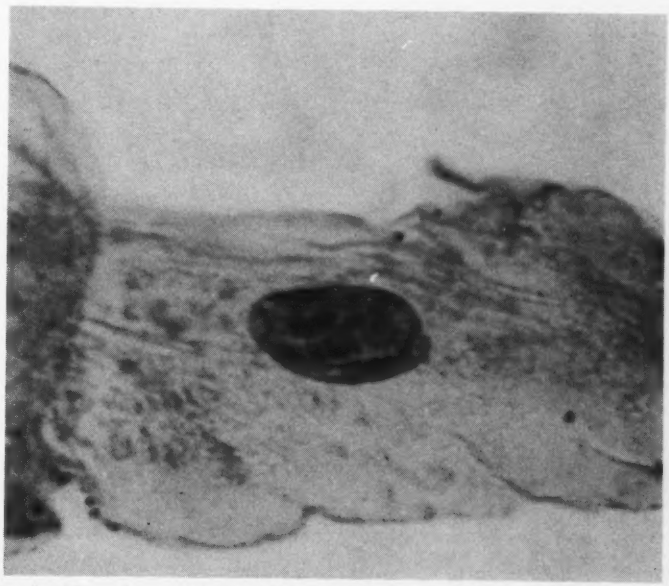

FIG. 2. Photomicrograph of buccal smear showing characteristic male arrangement of nuclear chromatin (Papanicolaou stain, × 2000).

this as a cause of amenorrhea, for its treatment with cortisone is one of the most satisfactory therapeutic efforts in the entire field of gynecological organotherapy (Jones & Jones, 1954).

There remains for consideration three principal varieties of male hermaphroditism. All patients considered have been reared as women and have had amenorrhea as an important clinical feature.

1. Gonadal aplasia.
2. Male hermaphroditism with ambiguous external genitalia and eunuchoid features.
3. Male hermaphroditism with feminine external genitalia and habitus.

Gonadal Aplasia

All patients with this syndrome exhibit amenorrhea and sexual infantilism. In a typical patient, the external genitalia are entirely feminine, but remain immature even in adulthood. In an interesting small subgroup, there may be phallic enlargement. The vagina is of small calibre and the uterus, palpated by rectum, is infantile. Examination of a vaginal smear shows no estrogenic stimulation. Puberty never appears and the pelvic findings remain infantile throughout life. There is never any breast development. Curiously enough at about the age of expected puberty, scanty pubic and vulvar hair, as well as axillary hair, usually appear.

One of the interesting general findings of this syndrome is the short stature. These patients seldom attain a height of 5′ although an occasional patient of average height has been described. One might comment that although the stunted growth seems to be an invariable accompaniment of gonadal aplasia, nevertheless, this feature cannot be explained on the absence of gonadal development for patients who are castrated in infancy grow tall and develop eunuchoid proportions. It, therefore, must be assumed that the stunting of the growth is a part of the general picture of anomalous development. It is not unusual to have a considerable number of associated somatic malformations. A webbed neck is a characteristic feature in approximately one-third of all patients and was one of the features described by Turner in 1938 when he delineated the clinical features of this disorder, known for many years as "ovarian agenesis" (fig. 3).

Fig. 3. Photograph of patient with gonadal aplasia. Note short stature and characteristic webbed neck. (Jones, Jr., H. W. and Scott, W. W., "Hermaphroditism, Genital Anomalies and Related Endocrine Disorders," Williams & Wilkins Co., Baltimore, Md., 1958.)

An important finding necessary to the diagnosis of gonadal aplasia is an elevation of the urinary pituitary gonadotrophin excretion, FSH. This, of course, would not be expected to be elevated until the patient had passed the age of puberty. However, above the age of 15 years, gonadal aplasia cannot be considered as a diagnosis unless the urinary FSH value is in excess of 50 m. u./24 hr.

One of the most revealing recent laboratory

findings in this syndrome was the discovery that a large proportion of these patients show a male chromosomal arrangement. The biologic significance and background of this discovery cannot be discussed at this time, but suffice to say that it is a finding entirely consistent with the brilliant work of Alfred Jost and other experimental embryologists who have demonstrated that feminine development of the internal and external genitalia invariably occurs in the absence of a normally functioning male gonad. Except for the maldevelopment of the gonad, these patients would have been men.

Laparotomy has uniformly demonstrated Mullerian ducts, perfectly developed although quite immature. The uterus is very small, the tubes likewise. The center of interest is the gonads. Instead of being normally developed, they are represented only by a very primitive streak visible grossly at operation (fig. 4). The microscopic examination of the excised genital ridge does not show any structure characteristic of the ovary. There is usually abundant wavy stroma with complete absence of germ cells. In short, there is aplasia or agenesis of the sex gland.

All patients with gonadal aplasia are reared as girls and women because of the entirely feminine configuration of the internal and external genitalia. There is, of course, no reason to consider a change in the social status even though the chromosomal arrangement may be male. In fact, if one could diagnose such patients at birth, they should be reared as girls and women in spite of the embryonic misadventure.

There is no known treatment that will stimulate the aplastic gonad to further development or function so that endocrine treatment of the disorder is confined entirely to replacement therapy by estrogen. This may be conveniently carried out with a daily dose of 1.0 mg. of stilbestrol or equivalent oral estrogen for an indefinite period of time. This will cause development of the sexual end organs such as the breast, labia, vagina, uterus and tubes. After development to a state of advanced adolescence, it is important to administer the estrogen in a cyclic manner to produce cyclic uterine bleeding. As an alternate and somewhat more controllable treatment, the estrogen may be continued indefinitely in daily doses and cyclic pseudomenstruation induced by periodically administering progesterone either orally or by intramuscular injection.

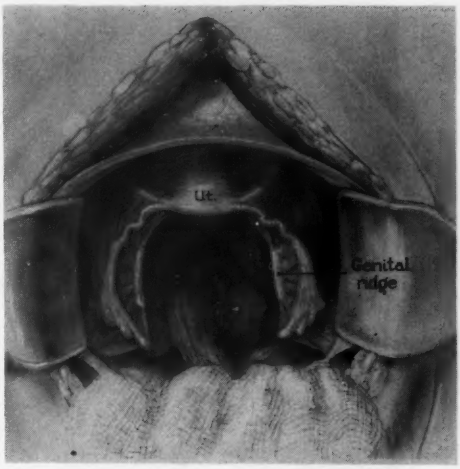

FIG. 4. Sketch of operative findings of patient with gonadal aplasia. (Jones, Jr., H. W. and Scott, W. W., "Hermaphroditism, Genital Anomalies and Related Endocrine Disorders," Williams & Wilkins Co., Baltimore, Md., 1958.)

MALE HERMAPHRODITISM WITH AMBIGUOUS EXTERNAL GENITALIA AND EUNUCHOID FEATURES

Primary amenorrhea is likewise an invariable finding in these patients. The external genitalia of this group are subject to considerable variation and the sex of rearing encountered has depended upon the judgment of all concerned at the time of birth. Obviously, for purposes under discussion, only those individuals who have been considered girls and reared as such will be taken into account at this time (fig. 5). This means that the external genitalia of this group tend to be more feminine than masculine

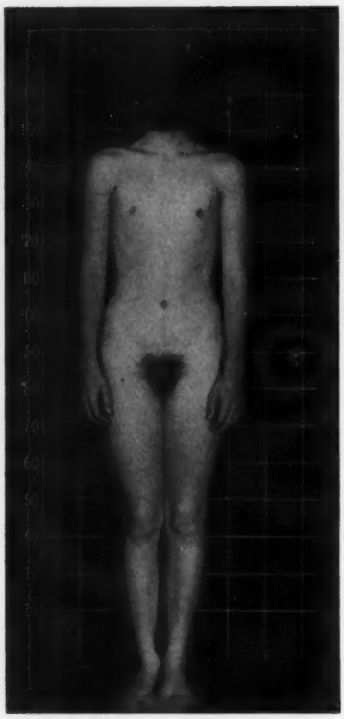

FIG. 5. Photograph of male hermaphrodite whose complaint was primary amenorrhea. (Jones, Jr., H. W. and Scott, W. W., "Hermaphroditism, Genital Anomalies and Related Endocrine Disorders," Williams & Wilkins Co., Baltimore, Md., 1958.)

although in none of this group are the external genitalia entirely feminine. There may be more or less fusion of the scrotolabial folds so that the external orifice of the vagina may be obscured. In all cases there is a vagina, but in some instances this may be rather shallow and in other instances it may be of normal depth. The phallus is enlarged to varying degrees (fig. 6). The internal genitalia are also subject to considerable variation and in approximately one-half of such patients the Mullerian ducts are completely inhibited during intrauterine life so that there is no uterus or Fallopian tubes. On the other hand, some patients will have well developed uteri and tubes although in these instances they are generally quite under-developed (fig. 7).

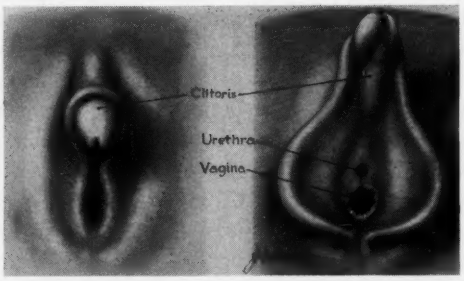

FIG. 6. Sketch of external genitalia of male hermaphrodite whose complaint was primary amenorrhea. (Jones, Jr., H. W. and Scott, W. W., "Hermaphroditism, Genital Anomalies and Related Endocrine Disorders," Williams & Wilkins Co., Baltimore, Md., 1958.)

As a general rule, patients in this group tend to masculinize at puberty. This is manifest in a negative way by the failure of breast development or other evidences of feminine secondary sex characteristics. In a positive way, masculinization may be indicated by hirsutism, lowering of the voice and increased size of the phallus. As already mentioned there is no sign of menstruation. The tendency to the development of masculine secondary sex characteristics is often a distressing feature. In order to prevent these undesirable phenomena, in patients reared as girls, it is necessary to predict, before puberty, the expected type of secondary sex characteristics which will develop in individuals whose external genitalia show some abnormalities of formation. As a rule of thumb, it has been found that *male hermaphrodites with entirely feminine external genitalia tend to feminize at puberty*, as will be discussed in the next section. However, *if the genitalia are at all ambiguous, the probabilities are that the patient will virilize.*

Inguinal hernias are not uncommon and in about half of the twenty odd such cases observed by the author, the mistake in sex identification was discovered by a surgeon during an operation for what seemed like an ordinary inguinal hernia.

Patients with well developed Mullerian structures, that is: uterus and tubes, have no special clinical or endocrinological findings which sepa-

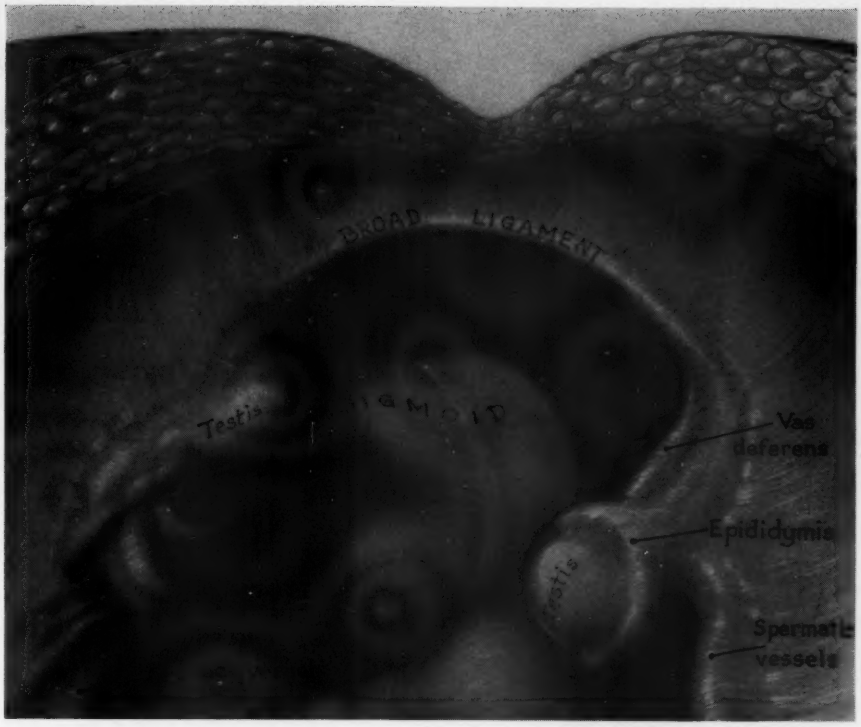

FIG. 7. Sketch of operative findings of male hermaphrodite reared as a woman. (Jones, Jr., H. W. and Scott, W. W., "Hermaphroditism, Genital Anomalies and Related Endocrine Disorders," Williams & Wilkins Co., Baltimore, Md., 1958.)

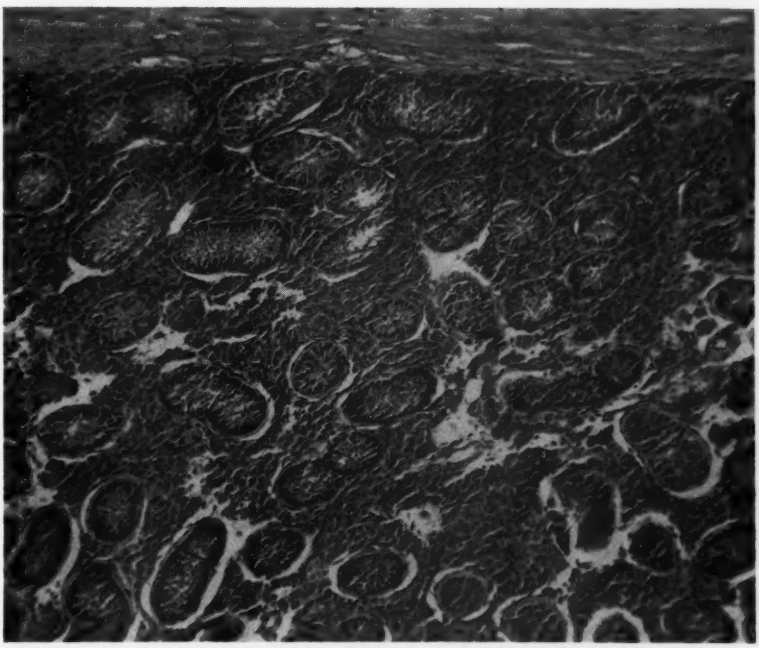

FIG. 8. Photomicrograph of testis of male hermaphrodite reared as a woman. (H & E, × 100) (Jones, Jr., H. W. and Scott, W. W., "Hermaphroditism, Genital Anomalies and Related Endocrine Disorders," Williams & Wilkins Co., Baltimore, Md., 1958.)

FIG. 9. Photograph of patient with testicular feminization syndrome. Her complaint was fatigability and amenorrhea. She was happily married. (Jones, Jr., H. W. and Scott, W. W., "Hermaphroditism, Genital Anomalies and Related Endocrine Disorders," Williams & Wilkins Co., Baltimore, Md., 1958.)

rate them from the group of male hermaphrodites without such organs. The presence or absence of the uterus, however, is of considerable theoretic and practical importance for its presence provides the possibility of cyclic uterine bleeding with the administration of estrogen.

All of these patients have cryptorchid testes. Microscopic examination of these organs show few, if any, spermatogenic cells. There is hyalinization of the basement membrane of the tubules and in some instances this process involves almost the entire structure. There is also apparent hyperplasia of the interstitial cells (fig. 8). It is apparent that these are abnormally developed testes which, in turn, were responsible for the maldevelopment of the external genitalia which, in their turn, caused the mistake in sex identification.

Treatment consists of the surgical removal of the abnormal testes because of their predilection for malignant change and their undesirable virilizing influence. This is preferably done before puberty, but, in any event, as soon as the diagnosis is made. Removal of the enlarged clitoris and lengthening of the vagina may be necessary. Exogenous estrogen may be given to cause feminine development of the secondary sex characteristics. In those patients who have uteri, cyclic estrogen and progesterone may be administered to cause cyclic uterine bleeding.

MALE HERMAPHRODITISM WITH FEMININE EXTERNAL GENITALIA AND HABITUS

(*Testicular Feminization Syndrome*) (*fig. 9*)

One of the most arresting syndromes in intersexuality is that of the genetic male with testes who resembles a normal appearing woman with excellent breast development. A number of authors have described such cases and Morris (1953) was able to collect 76 cases and report 2 new ones. However, this syndrome is apparently more frequent than would be indicated by this number of cases for the author has encountered seven such individuals in the last two years.

Patients complaining of primary amenorrhea should be suspected of this disorder. Many of these patients are so entirely feminine that it is not unusual for their only complaint to be that of amenorrhea. From the psycho-sexual point of view, these patients are entirely feminine. Usually there has been no suspicion that they are not genetic females. Several have been successfully married with quite normal sexual relations.

There is a strong familial predisposition to this syndrome and there are in the literature no less than 21 individuals who had sisters or aunts with clinical histories suggestive of a similar disorder.

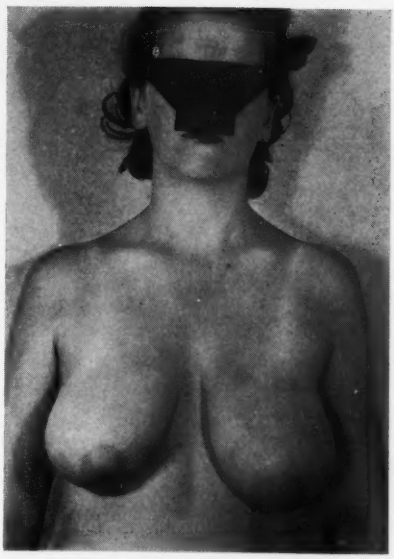

FIG. 10. Photograph of patient with testicular feminization syndrome showing adequacy of breast development.

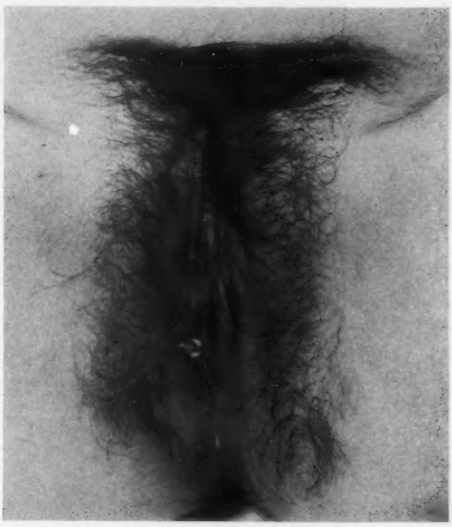

FIG. 11. Photograph of external genitalia of patient with testicular feminization syndrome.

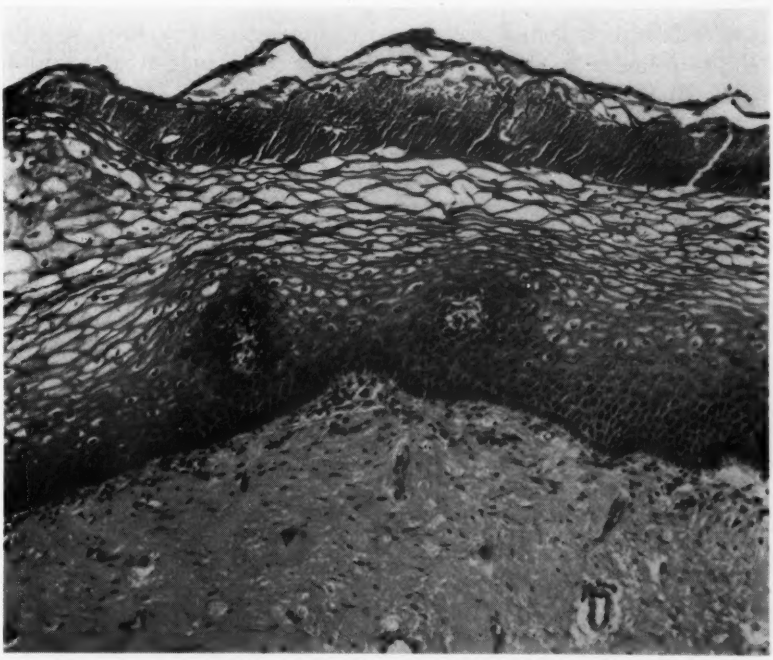

FIG. 12. Photomicrograph of vaginal mucosa of patient with testicular feminization. The mucosa was highly estrogenized although the patient's only gonad was a testicle (H & E, × 150).

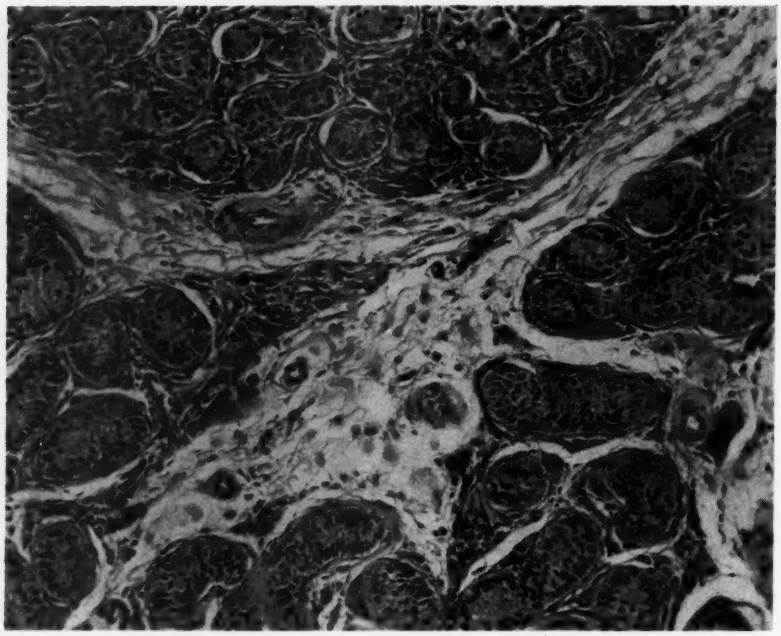

FIG. 13. Photomicrograph of testis of woman with testicular feminization syndrome. (H & E, × 150)

The patients have an entirely normal feminine habitus with normal feminine fat deposits. The breasts develop at puberty and there is often a tendency for over-development although the nipples may be juvenile (fig. 10). One of the interesting features of the disorder is the absence or scanty amount of axillary and pubic hair in the majority of cases. The hair on the head is that of a normal woman without temporal recession and facial hair is absent. The external genitalia are entirely feminine. The clitoris is normal or small (fig. 11). The vagina ends blindly, but is usually adequate for marital relations (fig. 12). There is absence of internal genitalia except for rudimentary uterine and other anlage including Fallopian tubes.

Gonads consist of testes with a very characteristic appearance on microscopic examination. The seminiferous tubules contain no spermatogenic cells, but the Sertoli cells are extremely well developed and contain a large amount of fat (fig. 13).

Hormone assays on a limited number of such patients suggest that these testes produce both estrogens and androgens but with a predominance of estrogens. After removal of the gonads both the urinary 17-ketosteroid excretion and the estrogens drop, indicating that the testes are the site of the excessive steroid production.

Since there is no uterus in any of these patients, the possibility of inducing cyclic bleeding by exogenous estrogen does not exist. One of the principal features of treatment is the insistence that the gonads be surgically removed because of the predilection for the development of seminomas in a small but significant group of patients. Continuous exogenous estrogen is necessary for an indefinite period.

SUMMARY

Male intersexuality may be an important unsuspected although infrequent cause of amenorrhea in patients who are reared as women. Gonadal aplasia, male hermaphroditism with ambiguous external genitalia and male hermaphroditism with entirely feminine external

genitalia all may be represented by patients whose principal and only complaint is amenorrhea. The clinical recognition, as well as the gynecological management, of these patients is discussed.

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BRONCHOLITHIASIS

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While the incidence of pulmonary calcifications has been found to be as high as 50% in some chest surveys,[15] a survey of the medical literature reveals the occurrence of broncholithiasis to be exceptionally low. Since the first detailed description of this condition appeared in 1600 [A.D.] by Schenk A. Grafenberg[2] In which he described 17 cases from the literature and 6 of his own, sporadic cases have appeared in the literature. By the latter part of 1946 Zahn[5] was able to find only 71 cases, adding another of his own. This was one of 4,000 cases of tuberculosis seen at the Fitzsimmons General Hospital in 1945. Stivelman[12] in 1928 reported one instance in 5,000 cases of tuberculosis and Pritchard[3] in 1923 reported two instances in 7,000 cases. Since then, three large series have been reported. Rabin[16] from the Mount Sinai Hospital 83 cases, Schmidt[1] from the Mayo Clinic 41 cases, and Groves and Effler[15] of the Cleveland Clinic 21 cases. At present about 300 cases have been reported in the literature, but this incidence is probably much lower than the actual occurrence of this disease, since many cases may be discovered by a heightened interest and increased awareness of this condition.

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PATHOLOGIC AND ETIOLOGIC FACTORS

The mechanism for the deposition of calcium in tissues has never been definitely established, but in chronically inflamed or necrotic tissues, changes in the pH with minimal carbon dioxide production and an increased alkaline reaction are thought to predispose to the deposition of calcium salts. These salts have a composition similar to bone. In fact, as pointed out by Poulalion,[20] broncholiths may be cartilagenous, calcareous or osseous. Wells[21] has pointed out the close analogy between calcification and ossification, and McPhedran[22] demonstrated this in the lungs. Wells quotes Poscharissky[27] as having examined 28 stones and finding bone in 17 (60%). One of our cases showed definite bone formation in a broncholith (Case 8). In some of the pulmonary calcifications tubercle bacilli have been found, and in others histoplasma capsulatum has been found.[18] In two of our cases tubercle bacilli were discovered in the broncholiths, and in another a careful search for histoplasma capsulatum revealed no evidence of this fungus, although the patient had a positive histoplasmin and negative tuberculin skin test.

Broncholithiasis may originate in three areas: 1), in the lumen of the bronchus,[26] 2), in the bronchial wall, 3), outside the bronchus in the tracheobronchial lymph nodes which erode through the wall of the bronchus. When calcium deposits

are formed in the lumen of the bronchus, bronchial secretions may act as a nucleus for the broncholith,[26] but this is the most infrequent type of this condition in the tracheobronchial tree. Occasionally, senile calcification or calcification associated with infection of the elastic part of the bronchi with sequestration may result in bronchial calculi. This condition is known as tracheopathia osteoplastica, the largest series of which (seven cases), were reported by Carr and Olsen[13] in 1954. The majority of broncholiths are due to calcification of the tracheobronchial nodes, secondary to an antecedent pulmonary infection. These erode the bronchial wall gaining partial or complete entrance into the lumen and acting like a foreign body with symptoms of obstruction of the bronchial tree drained by the affected bronchus.

Factors which precipitate migration of calcifications are unknown, although Head and Moen[8] suggest non-tuberculous infection as a cause. Auerbach[9] suggests erosion of the bronchial wall as a result of continuous respiratory motion. Undoubtedly, cough plays an important part in causing the broncholith to rupture into the lumen of the bronchus.

Calcification of the lungs and tracheobronchial nodes have been observed in: 1) inflammatory conditions, 2) tumors or cysts and infarcts, and 3) metastatic calcification which is due to deposition of calcium associated with hypercalcemia as in hyperparathyroidism, renal rickets, multiple myeloma, etc. There are no cases of broncholithiasis which are said to have occurred secondary to metastatic calcification. Of the infections, the granulomatous diseases are the most frequent cause, tuberculosis being the most common etiologic agent, particularly in the East. In the Mississippi Valley histoplasmosis is a more common cause, while coccidioidomycosis may be found to be the cause in the Far West. Other infections such as actinomycosis, pulmonary abscess, and bronchopneumonia may occasionally be the cause of broncholithiasis. The nodes most frequently involved in this condition are those: 1) occurring at the bifurcation of the trachea along the right main bronchus, 2) the middle lobe, 3) the anterior segment of the upper lobes, 4) the apical posterior segment of the left upper lobe, the left being less frequently involved than the right. The size of the broncholiths vary from that of a granule to one weighing[25] 139 gr. (9 gm.). They are greyish white, irregular, hard or putty-like, may be black due to anthracosis, yellow, and in one of our cases was orange-colored.

A node need not perforate entirely through the wall of the bronchus, but may be imbedded in its wall and covered only by granulation tissue, causing a narrowing of the lumen of the bronchus with signs of obstruction such as, atelectasis (as in Case II), recurrent pneumonitis (as in Case VI and VIII), bronchiectasis or lung abscess. When the stone perforates the bronchial wall, hemoptysis is likely to accompany the perforation. The disease rarely occurs in childhood because it takes several years for the lymph nodes responsible for the disease to calcify. However, we have seen one such case, of a child aged 4, in our series. Most of the cases reported, however, had their onset after age 40.

Symptoms

The symptoms of this condition result from an irritation of the broncholith of the tracheobronchial tree. The severity of the symptoms depends on the size and location of the broncholith. Cough is the most frequent symptom ranging from a mild cough associated with the raising of a small broncholith to severe spasmodic cough, paroxysmal in character called bronchial colic. The latter is frequently associated with intense substernal or parasternal pain which radiates laterally to the axillary region on both sides. Although cough occurs in almost all the cases, occasionally the stone may be silent.

Hemoptysis is the next most frequent symptom and occurs in 75–85% of the cases. Massive hemoptysis occurs occasionally, and may be present in as high as 30 per cent in some of the

series reported. At times, transfusions have been required and an occasional case may even result in death. Fever occurs as a result of obstruction associated with infection of the lung, usually associated with chills, and often with pleural pain, or retrosternal or parasternal pain. The fever is not infrequently associated with a cessation of the cough when the bronchus is completely blocked. Occasionally the onset may occur as a pleural effusion or empyema which obscures the underlying cause of the disease.[1] A number of patients may have an asthmatoid wheeze, which may be severe enough to be indistinguishable from intractable asthma or may simulate recurrent asthma.

Physical Examination

The physical findings vary from negative findings to those of a collapsed lobe with atelectasis and displacement of the mediastinum toward the affected side and decrease of the intercostal spaces with an elevation of the diaphragm. A frequent finding, as in two of our cases, was ronchi, groans and squeaks over the entire chest, louder in the involved area and disappearing when the broncholith was removed or expectorated.

X-ray

In most cases, unless the history of expectoration of broncholiths has been obtained, the diagnosis may not be evident on the X-ray unless one suspects it. There may be evidence of an obstructive lesion with atelectasis which may be diagnosed as a carcinoma, as in two of our cases, or a diagnosis of pulmonary abscess, empyema, bronchiectasis, or tuberculosis may be made without suspecting the broncholith as the underlying cause of the disease. Schmidt[1] believes that the most diagnostic roentgenologic finding is the presence of a dense area of calcium deposit at the apex of a triangular portion of collapsed lung. The apex of the triangle is located at the mediastinum, the collapsed portion corresponding to a segment of a lobe. The area of calcification may be obscured by the heart, so that heavy exposure may be required to visualize it. Rabin[16] believes that the calcific deposit when situated directly at the location of the origin of the bronchus is diagnostically significant. However, sectional radiography, oblique and lateral films, and bronchograms, may be necessary to show the relationship of the calcific deposit to the obstructed lobe or bronchus. Bronchography may also be useful in delineating an obstruction to the flow of radiopaque material at the site of the calcific deposit. The presence of a calcified node obstructing a bronchus, however, is not sufficient evidence to make the diagnosis, since the calcified node must be demonstrated in the lumen of the bronchus.

Bronchoscopy

Positive findings can be found on bronchoscopy in almost all cases, but only in about one-half can the broncholith be seen and removed, and occasionally it may be coughed up after bronchoscopy. In many cases the bronchus is found to be strictured or obstructed by granulation tissue, and not until biopsy of the area is the true cause discovered. The bronchoscopy findings are often suggestive of a tumor when the broncholith is obscured by granulation tissue and, in fact, carcinoma has been known to be associated with the disease in some cases. It is extremely important to biopsy the obstructed area, since the disease may be associated with bronchogenic carcinoma. At times the broncholith may be coughed up immediately after the bronchoscopy or several days later. The affected bronchus may be dilated when the broncholith can be seen but cannot be removed, whereupon the broncholith may be expectorated at a later date. When the condition is discovered relatively early, before considerable damage to the bronchus and lung has taken place, the bronchus may return to its normal state. If the broncholith is located in the bronchial tree, where it cannot be visualized, it is safest to explore this lesion, since a large

percentage of these lesions may turn out to be bronchogenic carcinoma. At the time of thoracotomy the broncholith may be found and the benign nature of the disease determined so that conservative rather than radical surgery would be indicated. After the broncholith has been extruded a residual stricture may remain resulting in bronchiectasis and recurrent pneumonitis. The obstructive pneumonitis which occurs consists of dilatation of the bronchi; the pulmonary alveoli and bronchi being filled with exudate and having varying degrees of fibrosis, depending on the length of time that the obstruction has been operative. This obstruction may simulate that caused by carcinoma of the lung and is, therefore, frequently mistaken for carcinoma.

Treatment

In non-operative cases the expectoration of numerous stones is still compatible with long life.[24] Some cases are on record as having lived for 22 years, while expectorating innumerable stones. In about 25 per cent of the cases stones may be removed through the bronchoscope. As noted under Bronchoscopic Examination, bronchoscopy is of value in the removal of stones or dilatation of bronchi so that the stones may be expectorated later.

It is the opinion of some observers[16] that conservative therapy is the treatment of choice because resection may be technically difficult and attendant with considerable hemorrhage at the time of operation, often requiring the removal of much healthy tissue. Because of the inflammatory reaction at the site of the broncholith, what may begin as a segmental resection may end in a pneumonectomy. This latter view is not shared by us since, in the experience of one of us (O.C.B.), technical difficulties in operative procedures of the lung are increased in the presence of broncholithiasis but are not at all insurmountable. At times the diagnosis may be mistaken for tumor as in one of our cases, and a total pneumonectomy may be done where a lobectomy only, was indicated. Groves and Effler[15] advise prophylactic surgery consisting of the removal of major calcifications in the subcarinal and tracheal areas when these areas are thought to be the cause of the irritating cough, or are causing suggestive symptoms of obstruction. They believe that patients with major mediastinal calcification should be kept under surveillance and symptoms in the lower respiratory tract are strong indications for bronchoscopic examination.

In our opinion prophylactic surgery is not a good principle, since the multiple sites of origin of calcified areas would preclude the removal of all of them. Those that remain could readily be a source for further symptoms. Surgery, when contemplated therefore, should be aimed at the removal of the offending broncholith and such calcifications in the immediate area as can be removed with facility. Lung tissue which has been irreversibly damaged by the disease processes obviously must be resected.

Case Material

The first three cases are adults who had broncholithiasis associated with active tuberculosis, the fourth, a child with broncholithiasis with probable active tuberculosis, the fifth an adult with pneumonitis and residual bronchiectatic lesions and the sixth and eighth cases were examples of recurrent pneumonitis due to obstruction. The seventh case was an obstructive lesion simulating a carcinoma.

Case I. This patient was a 30-year old white male who was admitted to Eudowood Sanitarium on July 29, 1949. He developed active pulmonary tuberculosis just before admission. On admission his sputum was found positive for tubercle bacilli. His treatment thereafter consisted of streptomycin and P.A.S. and, subsequently, pneumoperitoneum, pneumothorax on the left, followed by pneumonolysis which was unsuccessful and, finally, a partial thoracoplasty on the left which was done in May 1951. His original disease consisted of a cavitary lesion in the left upper, his sputum failing to convert

to negative until after his thoracoplasty. X-ray showed evidence of calcification in the left hilus region. In November 1950 the patient expectorated a calcified nodule in which tubercle bacilli were demonstrated after the nodule was crushed and stained for acid-fast bacilli. His sputum had been negative for tubercle bacilli for six months previously, but he complained of wheezing in his left chest with considerable cough following an unsuccessful pneumonolysis in September 1950. It was thought at the time that he had tuberculous bronchial disease, since his sputum had become positive for tubercle bacilli both on smear and culture at the same time that he expectorated a broncholith. These findings were associated with considerable wheezing in his left chest. X-ray at the time showed no definite evidence of active disease.

Bronchoscopy in Feb., 1951, showed purulent material coming from his left upper lobe bronchus. This was found positive for tubercle bacilli. In April 1951 he again expectorated a calcified nodule which contained numerous tubercle bacilli. His sputum still showed tubercle bacilli occasionally. A thoracoplasty was done on the left side in May 1951 with the idea of doing a resection of the left upper lobe if his sputum remained positive. However, his sputum converted to negative after the operation and he expectorated no more broncholiths. He was discharged from the Sanitarium in September 1952 and has remained well since. The finding of tubercle bacilli in the broncholiths expectorated by this patient suggests the possibility of finding the etiologic agent in the extruded calcified material, if the material is examined for it.

Case II. W. B.—A 55 year old white female was admitted to Eudowood Sanitarium October 5, 1954, for the second time. The first admission was on October 1, 1939, with symptoms of pleurisy with effusion. She was discharged March 13, 1940, at which time her chest film was clear. She had previously been diagnosed as having tuberculosis in 1929, at which time she had expectorated a small amount of blood, but had a negative chest film. During 1939, her pleural fluid was aspirated twice. Tubercle bacilli were not found. In 1946 she developed a small parenchymal lesion, but was not seen by us again until the last admission. In the meantime, she had remained well until January 1951, when she developed a viral infection which cleared up in a couple of weeks, but two months later she had a small hemoptysis for which she was admitted to another hospital. At that time she was told there was a mass in her left upper lobe on x-ray, and bronchoscopy revealed the left upper lobe bronchus to be only partially visualized. It was filled with milky pus and seemed to be partially obstructed with granulation tissue. This material which was removed, was described by the pathologist as being granular and having no neoplastic cells. Apparently, it was not examined for tubercle bacilli. A resection of the left upper lobe was done and a diagnosis of tuberculoma was made. Following this operation she was returned home, but retained a persistent cough and profuse expectoration. In August 1954, she had another small hemoptysis and expectorated several broncholiths. In the history obtained by the bronchoscopist, the patient stated that she had coughed up several hard lumps and, at one time, a small "flying animal". She was again admitted to Eudowood Sanitarium in October of 1954, at which time her sputum was found positive for tubercle bacilli once more. She was put on antituberculous medication, her sputum converting to negative in January 1955, and has remained negative since. In 1956, the patient had a resection of a portion of her bladder for carcinoma with an uneventful recovery. In October 1957 she again expectorated two broncholiths, but, unfortunately, these were not obtained for study. Her x-ray 10-25-57, showed very little calcification in the hilus region on the left, with evidence of resection of several ribs and a contraction of her left lung due to the lobectomy done in 1951. In retrospect, the patient probably had a broncholith blocking the left upper lobe in 1951 with a dense lesion in

the left upper probably representing an atelectatic area. Subsequent expectoration of broncholith in 1954 was associated with an activation of pulmonary tuberculosis, from which she has since recovered on antituberculous medication.

Case III. J. D.—A 49 year old white female who developed tuberculosis in 1929, and was sent to the State Sanitarium. There she was found to have active tuberculosis with a positive sputum. Her x-ray showed scattered infiltration throughout the right lung and dense infiltration in the left upper. For the first four months after her admission she recalls expectorating numerous greyish-white broncholiths. She did not report these to her physician, because other patients told her that this was to be expected and was not unusual. She had had tuberculous laryngitis, which was cauterized while at the State Sanitarium. At the time of discharge after one year of hospitalization she was considered improved and her sputum was negative for tubercle bacilli. In May 1930 she entered the State Sanitarium Training School for Nurses, but developed active tuberculosis again, and was admitted to Mount Wilson in November 1932. The disease had become worse since her admission to the State Sanitarium, but improved on bed rest, and she was discharged in September 1933. In 1939 she was employed as a nurse at Eudowood, and apparently her health was fairly good until February 1940, when she began to expectorate and noted some small orange colored granules in her sputum. Her sputum at this time was found to be positive for tubercle bacilli, and one of these granules contained numerous tubercle bacilli. She was told by her doctor that this granule probably was calcified tissue. The x-ray at this time showed numerous calcified nodules in both upper lung fields, particularly in the left hilus region. A film taken 11-15-57, showed further calcification of an area in the right mid-lung region, which replaced the soft infiltrate present in this area in 1940. She has remained well since 1941. Bronchoscopy was done in 1940, and was reported as essentially negative, with a questionable bronchial ulcer on the right.

Case IV. A 4 year old child was seen on April 3, 1956, with a past history of having had an attack of nephritis one year before, characterized by vomiting, fever and bloody urine. She was hospitalized for ten days and made an uneventful recovery. However, in December 1955, she coughed up gritty material thought to have been numerous broncholiths. The patient was bronchoscoped and a slight inflammation of the tracheobronchial tree was found, but no other abnormality was seen The physical examination was normal. An x-ray of the chest showed an area of calcification in the parenchyma of the middle lobe and calcified glands in the right hilus. The lung fields were otherwise clear. No abnormalities were seen in the bronchogram. The patient had a highly positive tuberculin, and was put on antituberculous medication in March 1956, consisting of I.N.H. and Dihydrostreptomycin. In May 1957 the patient again coughed up some pearly, gritty material for a few days. The sputum cultures have been persistently negative for tubercle bacilli. The patient has continued on I.N.H. and Dihydrostreptomycin and has remained in good health.

Case V. Helen H., a white female aged 41. This patient is an example of an obstructive pneumonitis and bronchiectasis due to a broncholith. She was first seen on November 28, 1948. She gave a history of having been told that she had bronchiectasis several years before. Two weeks before she was seen she had begun to cough, expectorate and had bouts of fever. For the last two months she coughed up blood on several occasions and had pain in the left side of her chest. This pain was worse on coughing and on deep breathing. Temperature was 102.6 F. She was put on Penicillin 300,000 units daily. On November 29, 1948, during the night, the patient had a severe coughing attack and coughed up a sizeable broncholith about 1 cm. in diameter. X-ray at this time showed two calcified glands in the left mediastinum and an area of calcification in the parenchyma of the

lower lobe, obviously a Ghon tubercle. There was diffuse infiltration involving the greater portion of the left upper lobe with a shift of the mediastinum to the left. Bronchoscopy showed no inflammatory or neoplastic changes on December 21, 1948. There was slight hyperemia of the left main bronchus. X-ray in January 1949 showed the clouding of the left upper to have cleared considerably with only slight stringy infiltration in the left upper. She was considered much improved. Bronchogram done May 10, 1949, revealed bronchiectasis of the lingula with narrowing of the lingular bronchus. A resection of the lingula of the left upper lobe was therefore done on May 16, 1949, with an uneventful recovery. She has had no further hemoptysis and a survey film in 1956 showed no evidence of active disease. The patient was considered well.

Case VI. A 30 year old white male, seen in February 1957. He began to cough four or five months previously and brought up bits of calcified material in the sputum from time to time, varying from small pea-size granules to nodules 1½ cm. in size. Just before admission he developed fever and chilly sensations, with a temperature up to 101 degrees Fahrenheit, and he began to complain of rather severe chest pains radiating from the substernal region to both axillae. He also had pain in his right chest, which was worse on deep breathing. Physical examination revealed marked wheezing at the right base posteriorly and laterally. The wheezing was less marked in the left lung. Patient was put on Tetracycline 250 mg. q.i.d., his temperature and symptoms clearing up in several days, although he still had numerous ronchi at his right base when he was seen several days later. He had another bout of fever two months later, similar to the first attack. This again subsided in several days after being given Tetracycline. Tuberculin, coccidioidin and blastomycin skin tests were negative but the histoplasmin skin test was markedly positive. The broncholiths were examined for histoplasma capsulatum and other fungi, as well as tubercle bacilli, but no evidence of any organisms or fungi were found on smear or culture. Bronchoscopy on February 13, 1957, revealed granulation tissue between the upper and middle lobe bronchus and a narrowing of the right middle lobe bronchus. There was evidence of non-specific bronchitis involving both the right and left main stem bronchi. Immediately after the bronchoscopy the patient expectorated a broncholith. A bronchogram done on February 20, 1957, was entirely negative. Since the bronchoscopy the patient has been relatively asymptomatic and physical examination revealed his lungs to be clear on November 20, 1957. X-ray was negative with evidence of minimal calcification in both hilar regions. There were increased trunk markings at the right base at the time he had evidence of bronchial obstruction. A photograph of several of the broncholiths is shown below. The patient expectorated about 30 of these broncholiths over a period of several months.

Case VII. Referred by H. T. This is a white male, aged 55, with symptoms of cough, expectoration, and hemoptysis. Bronchoscopy revealed what appeared to be a tumor mass with infection in the left mainstem bronchus. Because of the apparent vascular nature of the mass, a biopsy was not done (1948). A bronchoscopic diagnosis of bronchogenic carcinoma was made. Roentgenogram of the chest revealed atelectatic changes in the left upper lobe. Bacteriological studies were negative. At operation the left lung was removed, and the specimen showed a broncholith partially protruding through the main stem bronchial wall adjacent to the left upper lobe orifice. There was no microscopic evidence of neoplasm. The patient made an uneventful recovery, but died about four years later from the effects of the impaired pulmonary function.

Case VIII. M. M.—This patient, a white female aged 40, was admitted to the Johns Hopkins Hospital on September 8, 1955. This case was reviewed through the courtesy of Dr. Donald F. Proctor and Dr. Martin L. Singewald. She gave a history of productive cough, which

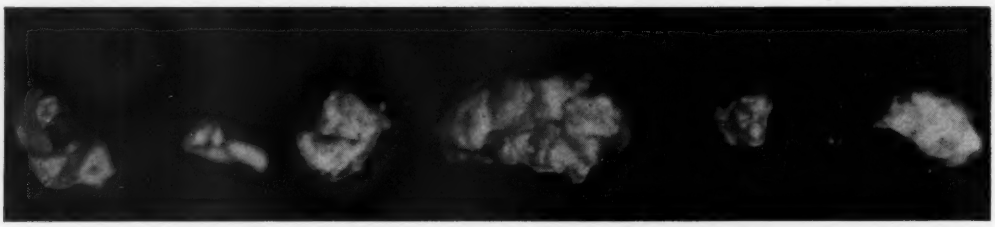

FIG. 1.

began several weeks before admission to the hospital. The cough had become so severe that she was unable to sleep at night. About a week before admission she began to have sharp pain on deep breathing on both sides of the lower chest posteriorly, associated with malaise and anorexia, with the pain gradually moving to the upper parasternal region on both sides.

On physical examination there were many groans and squeaks at both bases, but the patient was afebrile. She was put on Terramycin and Aminophyllin and gradually improved, being discharged from the hospital about a week later. X-rays showed increased markings in the hilus region on the right side. The diagnosis was acute tracheobronchitis.

She was well until next admission to the hospital on November 17, 1955, when she was admitted with a pleuritic type of pain in the third interspace on the right side, which was somewhat relieved by pressure over this area. Her cough had become troublesome again, and the interne noted that he had examined the patient on a number of occasions, and on two occasions her chest had numerous rhonci and wheezes, but on all other occasions was clear. She was again given Aminophyllin, Phenobarbital and Terramycin. An x-ray at this time showed a pneumonic process in the right hilus region involving the right upper lobe anterior segment, and also the right middle lobe. On November 28, 1955, bronchoscopic examination showed the right upper lobe almost occluded with thick white discharge coming from it. Biopsy was done, following which there was brisk bleeding. After a diagnosis of broncholithiasis was made from the biopsy material, it was decided to bronchoscope the patient again, and on December 2, 1955, bronchoscopy revealed a small stone in the right upper lobe bronchus. The operator could not dislodge the stone, and it was decided to leave it intact, and that the patient be bronchoscoped later. In the meantime, the patient gradually improved and the pneumonic area in the right upper and middle lobe slowly cleared.

On April 8, 1957, the patient was admitted to the hospital for bronchoscopy. The bronchoscopy showed moderate inflammation of the upper portion of the right main bronchus. There was some narrowing of the right bronchus in the neighborhood of the right middle lobe orifice, representing a partial stenosis of the right bronchus at this point; it was reduced to a diameter of 5 to 6 mm. The right upper lobe orifice appeared normal. Patient's x-ray appeared clear with surprisingly little calcification in either hilus region.

When last seen by her physician on November 15, 1957, she had negative chest findings. On microscopic examination this patient's broncholith showed evidence of necrotic bone formation.

DISCUSSION

We have reported eight cases of broncholithiasis, of which seven were probably due to tuberculosis and one to histoplasmosis. Three of these cases required an operative procedure for, or, as a result of, this condition and five did not require operation. There is some question as to whether patients with recurrent stones with evidence of obstructive pneumonitis should be operated upon prophylactically. Two of our patients who had evidence of obstructive pneu-

monitis were not operated upon prophylactically and appear to be in good health at this time, as are the other three nonoperative cases. This is consistent with our opinion that prophylactic surgery is not required for this condition, and as has been noted in previous reports, this condition may even be associated with long life. Of the three patients that were operated upon, one died of pulmonary insufficiency four years after his operation, one is still alive and well after an exacerbation of her tuberculosis two years after operation, and the other patient has fully recovered after a segmental resection for bronchiectasis, which was a residual of the broncholithiasis. Two of our cases were operated on for bronchogenic carcinoma by mistake. This error has been made frequently in the past. This condition must be suspected when there are signs of hemorrhage or pulmonary obstruction, by physical examination or x-ray, associated with hilar calcification. The diagnosis is confirmed when the patient either expectorates the broncholith, or it is found at bronchoscopy or operation. The usual age group has been from 30 years of age and up, although one of our patients was only 4 years of age at the onset of this condition. Study of the material in the broncholiths revealed Tubercule Bacilli in 2 of our cases, but no evidence of Histoplasmin Capsulatum in another probably due to histoplasmosis.

Summary and Conclusions

We have presented a brief resumé of the literature, and have added eight cases of broncholithiasis which have been proved by surgery, bronchoscopy or by the patient expectorating a broncholith. This is a rare condition which is probably much more common than the number of cases recorded, since many cases have been uncovered when the patient was either directly questioned about this condition or when it was considered in the differential diagnosis. It should be included in the differential diagnosis of any case of bronchial obstruction, pulmonary suppuration or hemorrhage associated with paroxysmal cough when the x-ray shows calcification. Curiously, in two of our cases the amount of calcification in the hilus was quite minimal. We feel that all of our cases were due to calcification of lymph nodes which perforated the bronchii, as has been noted in the majority of the cases reported in the past. The x-rays, symptoms and signs may simulate carcinoma of the lung, lung abscess, bronchiectasis, recurrent pneumonitis, or fungal infection. Special x-ray techniques such as laminography, lateral and oblique films, bronchograms and bronchoscopy, are important in establishing the diagnosis. The material in the broncholiths should be studied for evidence of infection since the etiologic agent may be found in them. Early diagnosis and treatment are important since the broncholith can be removed and residual lung damage may be prevented. A conservative approach is the treatment of choice, except where there is pulmonary obstruction with irreversible damage to the lung. Prophylactic surgery for this condition is not considered a good principle and the offending broncholiths should only be removed surgically when their exact location can be determined. It is technically impossible to remove all the calcification found at surgery and those that remain may just as readily be the source of further symptoms. The technical difficulties of an operative procedure in the presence of broncholithiasis should not deter one from contemplating surgery when it is indicated.

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This concludes the first section on the Church Home and Hospital. Additional articles will appear in the April Maryland State Medical Journal

CHURCH HOME AND HOSPITAL

Officers of the Board of Trustees of Church Home and Hospital are: the Rev. David C. Watson, President; the Rt. Rev. Harry Lee Doll, Vice-President; David W. Barton, Treasurer; Alexander Harvey, II, Secretary. Visitor to the Institution is the Rt. Rev. Noble C. Powell, Bishop of the Episcopal Diocese of Maryland.

ARTICLE OF INTEREST

ORGANIZATION AND OPERATION OF THE BOARD OF MEDICAL EXAMINERS OF MARYLAND

LEWIS P. GUNDRY, M.D.*

Article II†

The Board of Medical Examiners of Maryland is composed of eight members. Two examiners are elected each year for a term of four years by the Medical and Chirurgical Faculty of Maryland at their annual meeting.‡ According to Senate Bill 13 (Paragraph 118) "The examiners shall be physicians actually engaged in the practice of medicine and surgery in the State of Maryland and of recognized ability and integrity." The Board of Medical Examiners by provision of law meets "on the first Tuesday of June of each year for the purpose of organization." At this meeting the Board elects from its members a President, Vice-president and Secretary-Treasurer.

The President presides at meetings of the Board, represents the Board in conferences with the Medical Society and signs all licenses issued by the Board. The Vice-president presides in the absence of the President and assists with his executive duties. The Secretary-Treasurer keeps the minutes of the meetings of the Board; he also arranges all meetings and their agendas. He supervises all correspondence and signs all licenses issued by the Board. He is responsible for the financial business of the Board, signs checks and gives a financial report to the Medical and Chirurigcal Faculty. These three officers constitute the Executive Committee of the Board. They serve for a term of one year and may be re-elected.

The Executive Committee attends to the business of endorsement which necessitates almost monthly meetings. The entire Board meets about five or six times a year. The Secretary-Treasurer and the Board could not operate in an efficient manner if it were not for the Executive Secretary.§ The Executive Secretary is a full time employee of the Board. She supervises the office at 1215 Cathedral Street, takes care of routine correspondence, keeps the minutes of meetings, maintains a roster of physicians licensed by the Board, prepares licenses for signature and performs other tasks too numerous to mention. She has one full time assistant and several part time assistants when the work load is especially heavy.

The Board gives two written examinations each year. These examinations last four days. They begin on the third Tuesday in June and the second Tuesday in December. Eight three-hour examinations are given. The subjects are Anatomy, Medical Chemistry, Physiology, Therapeutics-Pharmacology, Practice of Medicine, Surgery, Obstetrics, Pathology-Bacteriology. Questions in Hygiene and Public Health are included in the examinations in Practice of Medicine and Therapeutics-Pharmacology. A grade of 75 per cent is required in each subject.

Each member of the Board of Medical Examiners is assigned one of the above subjects. He prepares the questions for the examination; is present when the examination is given and personally marks all examination books. By a previously determined date all marks of the eight examinations are sent to the State Board office. A few days later the Board meets to canvass the result of the examinations. The names of the candidates for licensure are unknown to the examiners since only a number appears on the examination book. It has never ceased to astound

* President, Board of Medical Examiners of Maryland.

† The first article was published in the January 1958 Maryland State Medical Journal, page 5.

‡ The Board of Medical Examiners is elected by the Medical Society in only two other States, Alabama and North Carolina.

§ The Executive Secretary is Mrs. Rose F. Barry who came to the Board in March, 1956. Before Mrs. Barry, Miss Hannah A. McCarthy served faithfully and efficiently for many years.

the writer that the eight examination marks for a candidate arrived at independently by eight examiners are almost always closely in agreement.

After the meetings for canvassing results, licenses are prepared for those who obtain a grade of 75 per cent or more in each subject. The less fortunate ones may take a reexamination in the subjects in which they have failed. This necessitates a waiting period of six months until the next regularly scheduled examination.

The Board also licenses physicians by endorsement of "proper certificates of proficiency and professional standing" (licenses) from other states and the District of Columbia; Maryland likewise endorses the certificates issued by the National Board of Medical Examiners. Maryland endorses the licenses of thirty-eight states, the District of Columbia and Puerto Rico. Whenever there are ten or more cadidates for license by endorsement the Executive Committee of the Board meets to review their credentials and personally interviews the candidates. These meetings take place about once every month. The fee for written examination is $35, the fee for licensure by endorsement is $50. Licensing of physicians who wish to practice in the State of Maryland is the most important function of the Board. The revocation of a license to practice medicine in Maryland is also of great importance. The subject of revocation will be discussed in detail in another article. The Board also investigates, through the Police Department, all reports of persons alleged to be practicing medicine without a license. It receives complaints against physicians. These may be passed along to the Medical Conduct Committee of the Medical Society or may be handled directly by the Board. In addition, it considers many problems connected with the practice of medicine in Maryland. To cope with the more difficult of these problems the advice of the Attorney General is frequently sought and followed. The Board in matters of great importance confers with the Council of the Medical and Chirurgical Faculty. Narcotics Law violations and alleged drug addiction on the part of a physician sometimes present perplexing problems. The local Bureau of Narcotics and the Attorney General are of great assistance in working out these problems.

The Board is represented annually at the Congress of Medical Education and Licensure. This is a meeting under the auspices of the Federation of State Medical Boards of the United States and the Council on Medical Education and Hospitals of the American Medical Association. The meeting lasts two or three days and is held in Chicago. The Maryland Board is represented by the Secretary and usually by one or two other members. A report of the meeting is given to the entire board. Much is gained by the formal and informal discussions with members of other State Boards at this meeting.

Members of the Board are compensated for meetings, for marking examination books and for travel and time when on official business for the Board. In addition the President and Vice-president receive a small monthly honorarium, and the Secretary-Treasurer a slightly larger one. Although the work is frequently time consuming and often frustrating, there is a deep satisfaction in the thought that the Board is maintaining a high standard of medical practice in Maryland and is protecting the public from unauthorized and improperly trained individuals.

1014 St. Paul Street
Baltimore 2, Maryland

The third article of this series
will be published in the April 1958 Maryland State Medical Journal.

Component Medical Societies

ALLEGANY-GARRETT COUNTY MEDICAL SOCIETY

LESLIE E. DAUGHERTY, M.D.

Journal Representative

Wills Mountain Sanitorium

The Wills Mountain Sanitorium was built in 1902, by local interests and was used first as a summer resort for two years and later leased to a Dr. Frey, in Washington, D. C., to be used as a Sanitorium for convalescent patients. Dr. James T. Johnson, Sr., of Cumberland, was local medical director. It was under operation two years and burned down in 1920, after several years of idleness.

Wills Mountain Sanitorium, Cumberland, Md.

1902–1920

Personals

Dr. George M. Simons, of Cumberland and Dr. Mildred Sheesley, Westernport, attended the 10th Annual Maryland Heart Association Meeting, held in November in Baltimore.

Dr. Earl Paul, recently addressed the Sacred Heart Hospital Alumnae Association, in Cumberland. His subject "Vascular Surgery" was accompanied by a motion picture.

Dr. Tom Van Strein, former Charles County Health Officer, has been appointed Health Officer for Allegany County. He is a native of Holland and a graduate in Public Health of the Johns Hopkins University. He succeeds Dr. Winter R. Frantz.

A Disaster Nursing Program was held in Allegany County during the months of October, November and December. One hundred and fifty-one nurses were given certificates for completing the course. The certificates were presented by Dr. Leslie E. Daugherty, Medical Director of Civil Defense in Allegany County. Dr. Daugherty recently addressed the Faculty of Fort Hill High School, in Cumberland, on "How School Teachers can best help in the Medical Civil Defense Program."

Sacred Heart Hospital, in Cumberland, has closed its obstetrical department as of January 1st, as a preparatory step toward the abandonment of the Institution within a two year period. Only three hundred babies were delivered in the past twelve months, making it an economic loss to continue.

BALTIMORE CITY MEDICAL SOCIETY

CONRAD ACTON, M.D.

Journal Representative

The regular monthly meeting of the Baltimore City Medical Society was called to order by the president, Dr. Frank J. Geraghty, at 8:40 P.M., Friday, November 1st. (Your representative was in San Francisco that week and is indebted to Dr. Lewis P. Gundry for notes on the activities. He was secretary of the Society for many years and is a past president. Could I write as he talks and as he saw, this column could take on new life and color.) About forty-five members were present. New members were elected. The well considered amendments to the Constitution were passed in toto without debate.

Dr. Grant E. Ward, chairman of the Nominating Committee, presented a slate of officers and delegates for the coming year. A call for nominations from the floor brought most outspokenly nomination for president and/or president-elect from one of our members dissatisfied with the status quo. The nominee promptly refused either position due to press of work.

After electing the slate presented by Dr. Ward, the president, Dr. Geraghty, turned the meeting

over to Dr. Robert E. Cooke, Professor of Pediatrics, The Johns Hopkins Medical School, moderator of the evening's Panel on "Pre-, Peri-, and Postnatal Considerations in Modern Preventive Medicine." The program, arranged by Dr. Harry C. Hull, chairman of the Program Committee, included Doctors George W. Anderson, obstetrician; David B. Clark, neurologist; Leon Eisenberg, psychiatrist; and James P. Miller, orthopedist, all of The Johns Hopkins Medical School. Dr. Cooke introduced the members and set the theme for the Panel: "Modern preventive pediatrics devotes itself to the prevention of serious, irreversible diseases." Illustrating his points with a slide, he declared that congenital defects are found in seven per cent of prematures and one per cent of full-term infants.

Dr. Clark estimated there are two million individuals in this country with birth handicaps. His slides regarding cerebral damage were clear and most interesting. Dr. Miller distinguished between traumatic and genetic, or truly congenital, disabilities. Trauma can be due either to uterine malposition or to labor mechanics. He outlined methods of correcting or helping the commoner ones, early recognition and correct diagnosis being essential. Dr. Eisenberg agreed with the basic premises that psychotherapy takes time and is used by all physicians, illustrating by the basic example that pediatric night calls are to reassure parents. He noted three basic reaction patterns in the newborn: a) the scream-and-yell type; b) the curl-up-and-quit type; and c) intermediate. Psychic trauma determines mental attitudes later on. Social experiences, such as family acceptance, are fundamental in this. Infants raised without parents, i.e., in isolation, have irreparable damage to the psyche with markedly difficult mental attitudes. A high percentage are criminals. Dr. Anderson spoke on obstetrical problems and epidemiological studies regarding congenital defects.

After a modest question and answer period, the members adjourned to the light and pleasant refreshment served by the Woman's Auxiliary.

* * *

At a regular meeting of the Executive Board, Tuesday, November 19, more than twenty items were on the agenda. The meeting lasted more than three hours. Many of the topics had numerous subdivisions. There were nine "grievances." Some trivial, like the lady who waited 'too long' in an outpatient department and objected to paying the hospital for talking to a foreign doctor who did not understand her. Others as serious as litigation between physicians and lawyers regarding fees and 'discounts.' The Board gave each one careful consideration and either reached a decision or called for more facts on which to make a judgment. The Board is most mindful of the far reaching public relations effects of its actions in these matters.

The State Faculty's Committee on Rural Health (nee Rural Medicine) had requested that all component Societies form local committees to work with it. Doubting the propriety of dabbling in "Rural" Health, President Geraghty had assigned an observer to attend the organization meeting November 14, 1957. The observer's report was that the activities of this State Committee corresponded with the City Society's Committee on. Public Education. His recommendation that this City committee, Dr. H. M. Robinson, Jr., chairman, act for the City Society in this matter, was approved.

A public relations firm wrote suggesting ten ways in which it could serve our Society. The project of employing our own public relations counsel was tabled until it is clear what is to be done at the State level.

Approval was given to a joint project between our Committee on Geriatrics, Dr. Herman Seidel, chairman, and the Mayor's Commission to Study Problems of the Aged in Baltimore. It is hoped that valuable statistics may be gathered in this way, at no expense to the Society.

Personal Health Information Cards, devised by the A.M.A. were forwarded to the component societies by the Faculty. They are similar to the "Immunization Registers" we remember from World War II. The A.M.A. will supply them to members for distribution to patients and most of the Board thought them a good idea. A modest number based on Society census was requisitioned for automatic issue.

BALTIMORE COUNTY MEDICAL ASSOCIATION

SAMUEL P. SCALIA, M.D.

Journal Representative

Rosewood State Training School was the scene of the monthly luncheon meeting of the Baltimore County Medical Association, on November 20, 1957.

It was announced that the Council of the Medical and Chirurgical Faculty has disapproved the Glenn L. Martin Company's closed-panel clinic. This is in agreement with the sentiments of our society. An over-all insurance plan is to be worked out with labor leaders in Maryland.

Our president, Dr. William A. Pillsbury, announced that Mr. William Wells, our legal adviser, had been sent to Chicago to represent us in the case of Dr. Robinson versus the American Medical Association and the State of Washington. This was deemed necessary by the A.M.A. because Dr. Robinson had attempted to gain membership in our society and, thus, we were directly involved.

The Nominating Committee, headed by Dr. Margaret Sherrard, submitted the following slate of officers: *President*, Dr. Clarence E. McWilliams; *Vice-President*, Dr. J. Morris Reese; *Secretary-Treasurer*, Dr. John E. Gessner. *Delegates:* Dr. Melvin B. Davis; Dr. Martin E. Strobel; Dr. William A. Pillsbury. *Alternates:* Dr. Frank T. Kasik; Dr. Louis Z. Dalmau; Dr. George S. M. Kieffer. No nominations were made from the floor. These nominees will be voted upon at the December meeting.

The Constitution has been revised and several important changes have been made. All members were issued a copy of the new Constitution. The membership unanimously accepted the changes.

Dr. Martin E. Strobel gave a report of the Delegates to the Medical and Chirurgical Faculty. He announced that there has been approval for the purchase of ground for a new Faculty building in Area 12 in Baltimore City. This will be near the Fifth Regiment Armory.

Dr. Charles F. O'Donnell presented a report of the Planning Committee. The Committee faces a multitude of problems in surveying the structure, function and administration of the Faculty. Dr. O'Donnell mentioned many of these problems. Numerous meetings are being held in an effort to solve these complexities. If any members of the county societies have any suggestions, the Planning Committee will be very happy to hear them.

The scientific part of the program was a film entitled "Children of the Wind." This film was developed at Rosewood through various grants. It is used in training employees for various departments. The film was very well done and a vote of commendation goes to the Rosewood staff for their efforts.

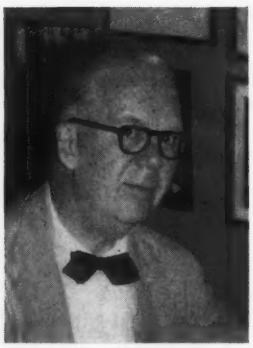
CHURCH HOME AND HOSPITAL

Financially Church Home and Hospital has strengthened its position over the last two decades. The Institution now has a net worth of some $6,000,000, no debts, and an "in-the-black" year-to-year operating record.

Necrology*

Joseph Earle Moore, M.D.

1892-1957



JOSEPH EARLE MOORE, M.D.

The death of Joseph Earle Moore on December 6, 1957 left a void which may never be filled in the lifetime of the majority of those who read this obituary. Dr. Moore was born July 9, 1892 in Philadelphia, Pennsylvania. He received his academic education at the University of Kansas, and graduated from the Johns Hopkins School of Medicine in 1916. After serving as a house officer for a period of one year at the Johns Hopkins Hospital, Dr. Moore entered the Medical Corps of the U. S. Army in which he served from April, 1917 to April, 1919. It was during this time that he first became interested in the venereal diseases, an interest which was to occupy so prominent a place in his future professional and scientific life. While in France he met his first wife, the former Miss Grace Barcley, who died in 1954.

Upon return to civilian life, Dr. Moore shortly entered private practice with Dr. Albert Keidel who had founded "Department L" at the Hopkins. In 1929, Dr. Moore became Physician-in-Charge of this department which, under his aegis, became the world's foremost venereal disease clinical and research center. Dr. Moore had the unusual faculty of attracting physicians with various skills, and the unusual ability to foster their research endeavors and interests which perhaps, more than anything else, made him the leader he was in the field. It is perhaps fitting to mention some of the foremost of these by name—Clark, Fleming, Nelson, Turner, Chesney, Crosby, Robinson, Heller, Vonderlehr, Farmer, Scott, and Eagle. The list includes at least six who subsequently became full professors as well as several subsequent leaders in the field of public health. Of perhaps equal importance was Dr. Moore's ability to attract and to train young physicians from both sides of the Atlantic, many of whom have subsequently become prominent in their own right.

Dr. Moore was a man who knew no prejudice with respect to creed or color, and I well recall that it was through his efforts that Negro physicians were first given the privilege of staff association with the Johns Hopkins Hospital. It was in Dr. Moore's clinic that the majority of the venereal disease control officers in World War II were trained and in his clinic also that the unique approach to the instruction of physicians in public health was instituted by having active participation on their part in the care of patients, a method of teaching of which he was particularly fond and which even now is being adapted to wider types of instruction in public health.

Dr. Moore was a tireless man of unbounded energy. He never grew old. Less than two weeks before his death, over a bit of cheer at his home, he told me with considerable enthusiasm of new plans for his clinic which had recently been expanded from a venereal disease to a general chronic disease environment.

Dr. Moore was a man heaped with honors. For many years he was Associate Professor of Medicine in the Johns Hopkins School of Medicine and a few months before his death achieved the signal honor of being the first practicing physician to be appointed full Professor in the department. Dr. Moore was for many years Adjunct Professor of Public Health Administration in the Johns Hopkins School of Hygiene and Public Health, an appointment at which he worked prodigiously.

Dr. Moore received the Medal of Merit for his work in venereal disease control during World War II. From 1940 to 1954 he was Chairman of the Subcommittee on Venereal Diseases of the National Research Council, during the latter four years of this tenure also being a member of the Committee on Medicine. From 1944 to 1946 he was Assistant Division Chief of the Committee on Medical Research. During the same period of time he was Chairman of

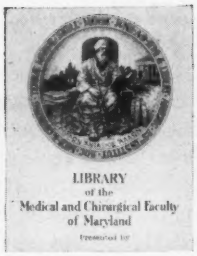
*A. S. Chalfant, M.D., "*Memoir Appointee.*"

the Syphilis Study Section, and a member of the Study Section on Experimental Therapeutics of the National Institutes of Health. He was consultant to the Surgeon General of the U. S. Army from 1943 to 1950. He was a Special Consultant to the United States Public Health Service from 1938 until the time of his death as well as consultant to the Maryland and New York State Health Departments and a member of a Special Advisory Committee to the Secretary of the Navy.

Dr. Moore was honorary Associate Professor of Medicine at Guy's Hospital in 1954. He was a member of Phi Beta Kappa and Sigma Xi, and of a number of learned societies including the Association of American Physicians, The American Society of Clinical Investigation, The American Clinical and Climatological Association, and The American Venereal Disease Association. He had corresponding membership in the Swedish, Danish, and Argentine Dermatologic Societies. Dr. Moore was Editor of the American Journal of Syphilis, Gonorrhea and Venereal Diseases from 1935 to 1954 and of the Journal of Chronic Disease from 1955 until his death. His publications include over 160 articles in the periodic medical literature as well as two authoritative text books, "The Modern Treatment of Syphilis" and "Pencillin in Syphilis."

Dr. Moore is survived by his second wife, the former Mrs. Mason Gieske. He is also survived by a host of younger physicians whom he trained and who loved him, as well as by untold thousands of people who never got, or were cured of, venereal disease because of his prodigious efforts.

Richard D. Hahn, M.D.
1422 Park Avenue
Baltimore 17, Maryland



Library

Louise D. C. King *Librarian*

"Books shall be thy companions; bookcases and shelves,
thy pleasure-nooks and gardens." *Ibn Tibbon*

YOUR TELEPHONE

We have many wonderful inventions and conveniences which our grandfathers never dreamed of, not the least of which is the telephone.

As I so often stop waiting on a reader to answer the insistent ring of the telephone, I think of the story my father used to tell. He tried repeatedly to obtain an interview with a prominent man, only to be told each time he was "in conference." Not to be downed, he left the hotel conference room, went down stairs, telephoned him and got him immediately. This illustrates very well that the telephone may be useful, not only as a first thought, but also as a court of last resort.

Do you use the telephone to your library as frequently as it may expedite your transactions for you?

There are many ways in which your telephone can assist you in your use of the library to save you time, some of which are listed below, others will occur to you as the exigencies of the occasion may dictate.

1. Check addresses, verify names, both of people and organizations.
2. Check dates of meetings, programs etc.
3. Verify references.
4. Ascertain if a certain volume is in the library.
5. Reserve books to be picked up later, saving you the time waiting to have them gotten out for you after you are here.
6. Request books and/or articles on a given topic, to be read here at your convenience, or picked up later to be used at home. Please ALWAYS STATE WHICH CATEGORY; it makes a difference in the number and type of books assembled.

When telephoning for information, you should be more detailed than if you are in the building and can be consulted. It is also well to remember that information is searched for you in the various indices, and that the information you desire may not be indexed as an entity, but only obtained by reading through many articles. *It is this latter phase of research that we are not equipped to give you.* It is very time consuming, and often requires more medical knowledge than your librarians can give you. There are many factors which influence our selection of material for you such as diagnosis, etiology, therapy (medical and/or surgical), general over-all picture, prognosis, control and the ever present bugbear of the librarian—statistics. When requesting verification of a reference, or the finding of an elusive or half forgotten article, PLEASE give us all the information you have, author, title, subject, journal, volume, page and year and if you know it, the source from which you obtained the reference originally. If you had ever spent hours searching for a recent article, only to find it appeared twenty years ago, you would appreciate the importance of the small details.

Your library is but dialing distance from your desk.

STATE OF MARYLAND DEPARTMENT OF HEALTH

MONTHLY COMMUNICABLE DISEASE REPORT

Case Reports Received during 4-week Period, January 3–30, 1958

	CHICKENPOX	DIPHTHERIA	GERMAN MEASLES	HEPATITIS, INFECT. AND SERUM	MEASLES	MENINGOCOCCAL INFECTIONS	MUMPS	POLIOMYELITIS, PARALYTIC	POLIOMYELITIS, NON-PARALYTIC	ROCKY MT. SPOTTED FEVER	STREP. SORE THROAT INCL. SCARLET FEVER	TYPHOID FEVER	WHOOPING COUGH	TUBERCULOSIS, RESPIRATORY	SYPHILIS, PRIMARY AND SECONDARY	GONORRHEA	OTHER DISEASES	DEATHS: Influenza and pneumonia
Total, 4 weeks																		
Local areas																		
Baltimore County	21	—	2	—	131	1	7	—	—	—	6	—	2	12	—	—	—	10
Anne Arundel	3	—	3	1	14	—	1	—	—	—	—	—	1	5	1	2	m-1	5
Howard	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—
Harford	1	—	—	—	—	—	—	—	—	—	—	—	—	2	2	—	—	1
Carroll	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	7
Frederick	9	—	1	—	—	—	—	—	—	—	1	—	—	3	—	—	—	2
Washington	—	—	—	—	—	1	—	—	—	—	—	—	—	5	—	—	—	2
Allegany	1	—	—	1	1	—	1	—	—	—	3	—	—	1	—	—	—	6
Garrett	—	—	—	—	—	—	—	—	—	—	3	—	—	—	—	—	—	1
Montgomery	37	—	4	2	45	1	4	—	—	—	13	—	1	6	—	4	m-1	2
Prince George's	6	—	2	—	15	—	4	—	—	—	4	—	1	15	1	4	—	6
Calvert	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	c-1	—
Charles	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Saint Mary's	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	4
Cecil	—	—	—	4	—	—	—	—	—	—	7	—	—	1	—	—	—	2
Kent	7	—	—	1	3	—	—	—	—	—	—	—	—	1	—	—	—	—
Queen Anne's	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2
Caroline	—	—	—	—	3	—	—	—	—	—	—	—	—	—	—	—	—	1
Talbot	1	—	—	—	—	—	—	—	—	—	—	—	—	1	—	5	—	4
Dorchester	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	—	1
Wicomico	—	—	1	—	2	—	2	—	—	—	1	—	—	—	—	4	—	5
Worcester	—	—	—	—	—	—	—	—	—	—	—	—	—	3	1	—	—	1
Somerset	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	2
Total, Counties	86	0	13	11	214	3	18	0	0	0	39	0	5	61	5	20		74
Baltimore City	85	0	26	2	1045	1	18	0	0	0	17	0	3	75	19	543		37
State																		
Jan. 3–30, 1958	171	1	39	13	1259	4	36	0	0	0	56	0	8	136	24	563		101
Same period 1957	232	1	15	6	57	2	201	0	0	0	74	0	22	131	22	530		63
5-year median	398	1	32	15	478	5	202	1		0	170	1	30	130	20	600		80
Cumulative totals																		
State																		
Year 1958 to date	209	1	49	14	1531	4	45	0	0	0	63	0	12	156	27	613		131
Same period 1957	300	1	19	6	62	3	239	0	0	0	81	0	23	168	26	759		78
5-year median	486	1	38	17	563	6	242	1		0	199	1	42	160	24	780		101

c = congenital syphilis.

m = meningitis other than meningococcal.

Woman's Auxiliary Medical and Chirurgical Faculty

MRS. HOMER ULRIC TODD, SR., *Auxiliary Editor*

NATIONAL AUXILIARY TO INCORPORATE

The National Auxiliary is to be incorporated at the advice of the Law Department of the American Medical Association, which is now preparing the necessary forms so that briefs may be presented and action taken at the 1958 Convention.

REPORT OF THE WOMAN'S AUXILIARY TO THE WASHINGTON COUNTY MEDICAL SOCIETY

MRS. J. G. WARDEN, *President*

We are happy to present the 1957 program of the Woman's Auxiliary to the Washington County Medical Society. We are, indeed, proud of this Auxiliary for many reasons but especially proud of their being 100 per cent organized.

February 13, 1957. Luncheon meeting. *Guests:* Mrs. Homer U. Todd, Mrs. A. E. Goldstein. Installation of officers 1957–58.

March 30, 1957. Doctor's Day. Cocktails and buffet supper.

May 21, 1957. Evening meeting. Dessert and games. (Proceeds added to our nurse's scholarship fund.)

July 1957. Annual picnic. Members and husbands.

October 1957. Second three-year nursing scholarship awarded. Acted as hostesses for Cardiac Seminar sponsored by local nursing groups and the Heart Association. Health kits (toothbrush, toothpaste, and comb) presented to each student at our Special School (mentally retarded) by our auxiliary.

November 7, 1957. Joint meeting with the Washington County Medical Society. Cocktails, dinner, speaker. Business meeting. Election of officers 1958–59: *President*, Mrs. Ross Cameron; *First Vice-President*, Mrs. Robert Conrad; *Second Vice-President*, Mrs. Archie Cohen; *Recording Secretary*, Mrs. David Boyer; *Corresponding Secretary*, Mrs. Maynard Bacon; *Treasurer*, Mrs. Richard Young; and *Parliamentarian*, Mrs. Frederick Graff.

Some time ago the Commission on Accreditation of Hospitals pointed out that the Medical Library at the Washington County Hospital was inadequate and the aid of the Washington County Medical Society was enlisted to bring it up to the required standards.

The Auxiliary began working with the staff library committee. All books and periodicals were catalogued and properly filed. Many books were badly in need of repair and were prepared for binding. The bindings were made at the State penal farm nearby and were paid for by the Woman's Auxiliary. The accession record was brought up-to-date, and the Auxiliary helped with many of the other details that were necessary to get the library on a "running basis." Several excellent volumes were purchased by the Auxiliary as memorials and placed in the library.

The Auxiliary maintains a special fund to care for the binding of books and periodicals.

REPORTS OF THE FOURTEENTH NATIONAL CONFERENCE OF STATE PRESIDENTS, PRESIDENTS-ELECT, AND NATIONAL COMMITTEE CHAIRMAN OF THE WOMAN'S AUXILIARY TO THE AMERICAN MEDICAL ASSOCIATION

MRS. E. RODERICK SHIPLEY, *President-Elect**

The Conference was set up to give everyone a short course in group dynamics. The meetings were under the leadership of Dr. Martin Chworowsky, Ph.D., Director of the Albert M. Greenfield Center for Human Relations, University of Pennsylvania.

Sunday evening, before the conference officially opened, numerous members who had previously

* The Conference was held in Chicago, Ill. October 21–22–23, 1957, with Headquarters at the Drake Hotel.

signed up for the evening, met with Dr. Chworowsky, who gave a very interesting and concise talk on working with your organization. The primary requisites for a good meeting were stressed. It was with this introduction that the conference got under way the next morning.

Monday morning a continental breakfast of coffee and buns was served in the Meeting Hall. Presentation of Officers and guests followed. Dr. Ernest B. Howard, Assistant Secretary of the American Medical Association, spoke to us, his topic being Medical Legislation. The most important item in this category is the proposed amendment to the Social Security Act, by the Congress, to provide free hospitalization to old age and survivors benefits. This is the Forand Bill H.R. 9467. This bill is A.F.L.-C.I.O. sponsored. Blue Cross—Blue Shield may endorse this bill outright or by equivocation.

The Heller Report—A Study of the A.M.A. is complete and under study by the A.M.A. Recommendations will be presented in Philadelphia in December.

Today's Health has a new editor, Jim Liston, who is on a full-time basis.

Coverage under the Social Security Act for physicians is a question that can lead to splintering and diverting the Medical Profession. This would be a disastrous move, as explained in the A.M.A. pamphlet "Which Way?"

Dr. Chworowsky recommends for your reading, "The Lonely Crowd" by David Riesman, "The American Teen Age," and "The Organization Man." We are victims of overconformity and these books bring out the dangerous trends that are starting or have started.

How can we have effective programs? It must be a joint endeavor or we fall short. We must make other persons feel our work is important. In this discourse on Human Relations we were coached in the mechanics of having a good discussion group.

Luncheon followed the morning session and our guest speaker was the President of the A.M.A., Dr. David Allman. He made a brief but interesting talk on the importance of the Auxiliary and the Medical Society as a joint endeavor.

The afternoon was spent in our assigned discussion groups where we put in practice all the principles that Dr. Chworowsky had been coaching previously. There was a coffee break in the middle of the afternoon in the French Room, where all the displays by the various States and organizations were exhibited. Your President, Mrs. Clayman, had for display the earrings which we are selling for A.M.E.F. Various Presidents from other states purchased whole cards to take home so that they might share in the project for their own Auxiliary. It was generally conveived to be an excellent way to raise these funds.

Monday evening a session on Program Demonstration was held in the Grand Ballroom. Visual aids were stressed at this meeting; slide lectures, movies and panel discussions were portrayed.

Tuesday was an entire day of group discussion spaced by a morning coffee break and luncheon with "Table Talk." Each table bore a label of some project of the Auxiliary such as Legislation, Mental Health, Recruitment, etc.

Mrs. Paul C. Craig, President of the Woman's Auxiliary to the American Medical Association, entertained that evening with a "Sherry Party" where we enjoyed delicious hors-d'oeuvres that were sent for the occaion by the Pennsylvania State Auxiliary. The surroundings were pleasant and the company most friendly—need more be said?

Wednesday, the third and last day of the Convention, was spent at 535 N. Dearborn Street, the headquarters of the A.M.A. Dr. George F. Lull, Secretary and General Manager of A.M.A., welcomed all most graciously. A series of the current A.M.A. films were shown for us in order that we might become familiar with them and be able to use one or more in our work at home.

A final talk on group dynamics was given by Dr. Chworowksy summarizing the roles played by participants in group discussions which he titled "Patterns for Progress."

Luncheon in the cafeteria was enjoyed by all before saying good-bye and going our separate way.

Chicago was grey, wet, warm, and humid, with clouds obscuring the tops of the tall buildings for almost all of the Conference; but, until one stepped outside it was all warm, light, and friendly. Although a very busy time, much food for thought and many good ideas were exchanged here and I came away feeling like a schoolgirl getting out for the summer vacation. I have had an experience that I am glad I did not miss.

COMING MEETINGS

MARCH 1958

BALTIMORE CITY MEDICAL SOCIETY

Speakers: Arthur L. Haskins, M.D., A. Edward Maumanee, M.D., Robert A. Robinson, M.D., Robert W. Buxton, M.D., Theodore E. Woodward, M.D., and Gilbert H. Mudge, M.D.

"Panel Discussion—What's New in Medicine"

Friday, March 7, 1958 8:30 p.m. 1211 Cathedral Street

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SOUTHEASTERN SURGICAL CONGRESS

March 10, 11, 12, 13, 1958 Lord Baltimore Hotel, Baltimore

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JOINT MEETING OF SECTION OF INTERNAL MEDICINE, B.C. M.S. AND THE MARYLAND SOCIETY OF INTERNAL MEDICINE

Speaker: Thomas W. Mattingly, Brig.-Gen., MC, USA, Chief, Cardiovascular Section and Cardiac Laboratory, Walter Reed Army Medical Center.

"Cardiovascular Manifestations of Pheochromocytoma"

Monday, March 17, 1958 8:15 p.m. 1211 Cathedral Street

All meetings are held in the Medical and Chirurgical Faculty Building, 1211 Cathedral Street, Baltimore, Maryland, unless otherwise designated.

APRIL 16, 17, and 18, 1958

ANNUAL MEETING OF MEDICAL AND CHIRURGICAL FACULTY

REMINDER—HOTEL ROOM RESERVATIONS

A block of rooms has been set aside at the Sheraton Belvedere Hotel, Charles and Chase Streets, Baltimore, for those attending the Annual Meeting of the Medical and Chirurgical Faculty. The Hotel will take your room reservations *now*. When making your reservation be sure to mention that you will be attending the Annual Meeting of the Faculty.

Annual Meeting

MEDICAL AND CHIRURGICAL FACULTY

APRIL 16, 17, and 18, 1958

The Alcazar, Baltimore

Plans for the coming Annual Meeting of the Medical and Chirurgical Faculty are almost complete, and the following program has been scheduled:

WEDNESDAY, APRIL 16, 1958

2:15 p.m. Dr. Theodore L. Badger, Boston
Dr. James H. Forsee, Washington, D. C. } *Panel Discussion on Medical and Surgical Treatment of Tuberculosis.*
Dr. Edmund G. Beacham, Baltimore
3:25 p.m. Dr. George Crile, Jr., Cleveland. (*J. M. T. Finney Fund Lecture.*)
4:05 p.m. Dr. Currier McEwen, New York City. "*Collagen Vascular Disease.*"

Wednesday Evening

8:30 p.m. Dr. Lester R. Dragstedt, Chicago. (*I. Ridgeway Trimble Fund Lecture.*).

THURSDAY, APRIL 17, 1958

9:30 a.m. Dr. Jacob H. Conn, Baltimore. "*Hypnosis.*"
10:00 a.m. Dr. Garfield G. Duncan, Philadelphia. "*Oral Diabetic Treatment.*"
10:50 a.m. *Election of Board of Medical Examiners.*
11:00 a.m. Dr. David Gitlin, Boston. "*Gamma Globulin.*"
11:40 a.m. Dr. Howard B. Sprague, Brookline, Mass. "*Cardiology Subject.*"
12:30 p.m. *Round Table Luncheon at the Park Plaza Hotel.*
2:30 p.m. Dr. Irving S. Wright, New York City. "*Cerebrovascular Disease.*"
3:10 p.m. Dr. John H. Moyer, Philadelphia. "*Diuril.*"
3:45 p.m. Dr. Brian Blades, Washington, D. C. "*Diaphragmatic Hernia.*"
4:15 p.m. Dr. John K. Frost, Baltimore. "*Cytology.*"

Thursday Evening

Presidential Dinner—Sheraton Belvedere Hotel

6:00 p.m. *Cocktails* (Courtesy of Baltimore City Medical Society).
7:00 p.m. *Dinner.*
8:15 p.m. Dr. David B. Allman, President, American Medical Association.

FRIDAY, APRIL 18, 1958

9:30 a.m. Dr. Robert T. Monroe, Boston. "*Geriatric Subject.*"
10:10 a.m. Dr. Robert P. Glover, Philadelphia. "*Surgery for Coronary Disease.*"
11:00 a.m. Dr. Sara M. Jordan, Boston. "*Gastroenterology Subject.*"
11:45 p.m. Dr. A. McGehee Harvey, Baltimore } *Clinical Pathological Conference*
Dr. Arnold R. Rich, Baltimore

The Woman's Auxiliary Luncheon will be held at the Sheraton Belvedere Hotel on Wednesday, April 16, 1958, and all doctors are urged to attend.

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BE SURE TO PLAN TO ATTEND THIS VERY INTERESTING AND EDUCATIONAL MEETING OF YOUR STATE SOCIETY

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The Council and House of Delegates will meet on Wednesday morning, April 16, 1958, and the second session of the House of Delegates will be held on Friday afternoon, April 18, 1958.

ANNUAL MEETING

WOMAN'S AUXILIARY TO THE MEDICAL AND CHIRURGICAL FACULTY

Sheraton Belvedere Hotel, Baltimore

Wednesday, April 16, 1958

9:30 a. m. Registration, Blue Room, Second floor
10:00 a. m. General Session, Blue Room
12:30 p. m. Luncheon, Main Ballroom

Wives of the members of the Medical and Chirurgical Faculty are invited to the General Session and luncheon. It is hoped that the doctors will attend the luncheon.

MRS. RAYMOND V. RANGLE, *Chairman*,
Convention Arrangements

AMERICAN TRUDEAU SOCIETY

POSTGRADUATE COURSE

"THE MEASUREMENT OF PULMONARY FUNCTION IN HEALTH AND DISEASE"

Sponsored by the Medical Schools of
Harvard University, Tufts University and Boston University
Harvard School of Public Health
Massachusetts Tuberculosis and Health League, and the
Massachusetts Trudeau Society

March 24–28, 1958

Tuition $75.00

Applications and more detailed information may be obtained from the Chairman of the course:

Edward J. Welch, M.D.
1101 Beacon Street
Brookline 46, Massachusetts

MARYLAND SOCIETY OF INTERNAL MEDICINE

The officers of the newly formed Maryland Society of Internal Medicine are as follows: *President:* Dr. Edward F. Cotter, Baltimore; *President Elect:* Dr. John H. Hornbaker, Hagerstown; *Secretary-Treasurer:* Dr. Katherine H. Borkovich, Baltimore.

The official location of the Society is: Suite 5-F, 11 East Chase Street, Baltimore 2, Maryland.